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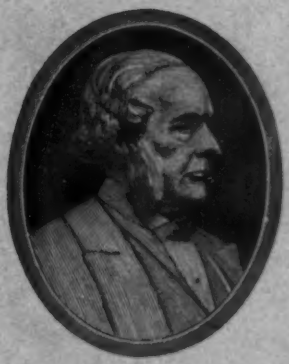

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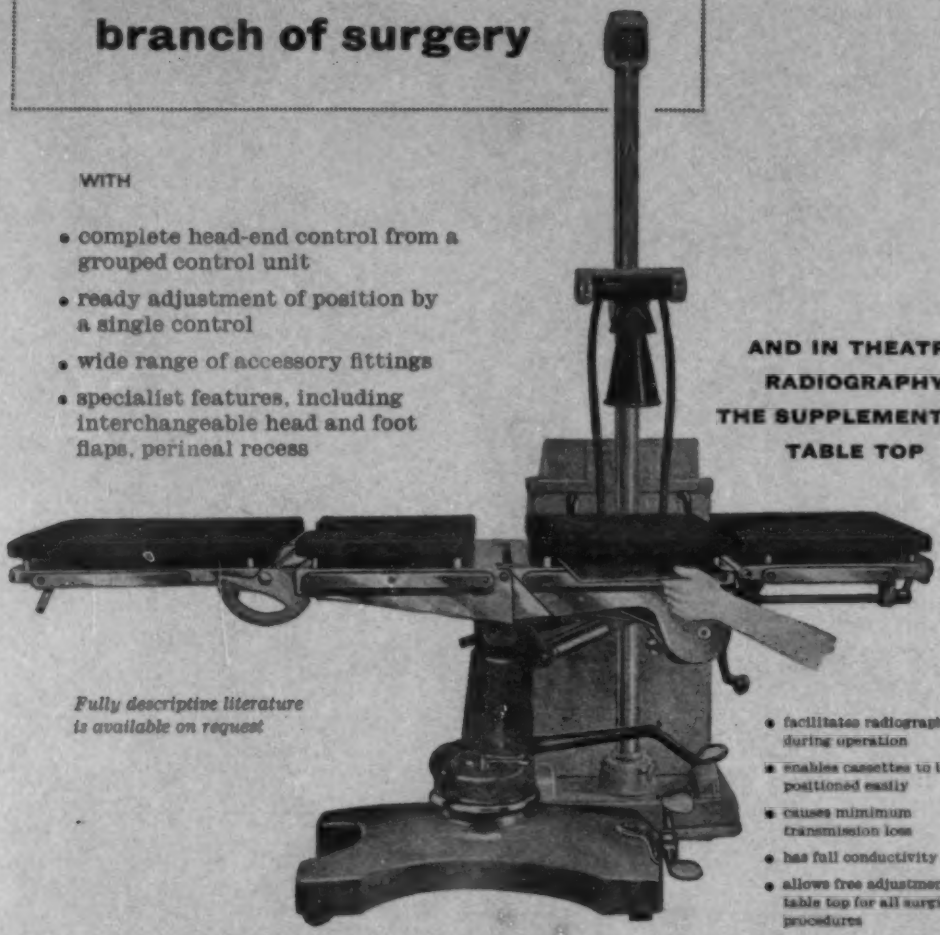

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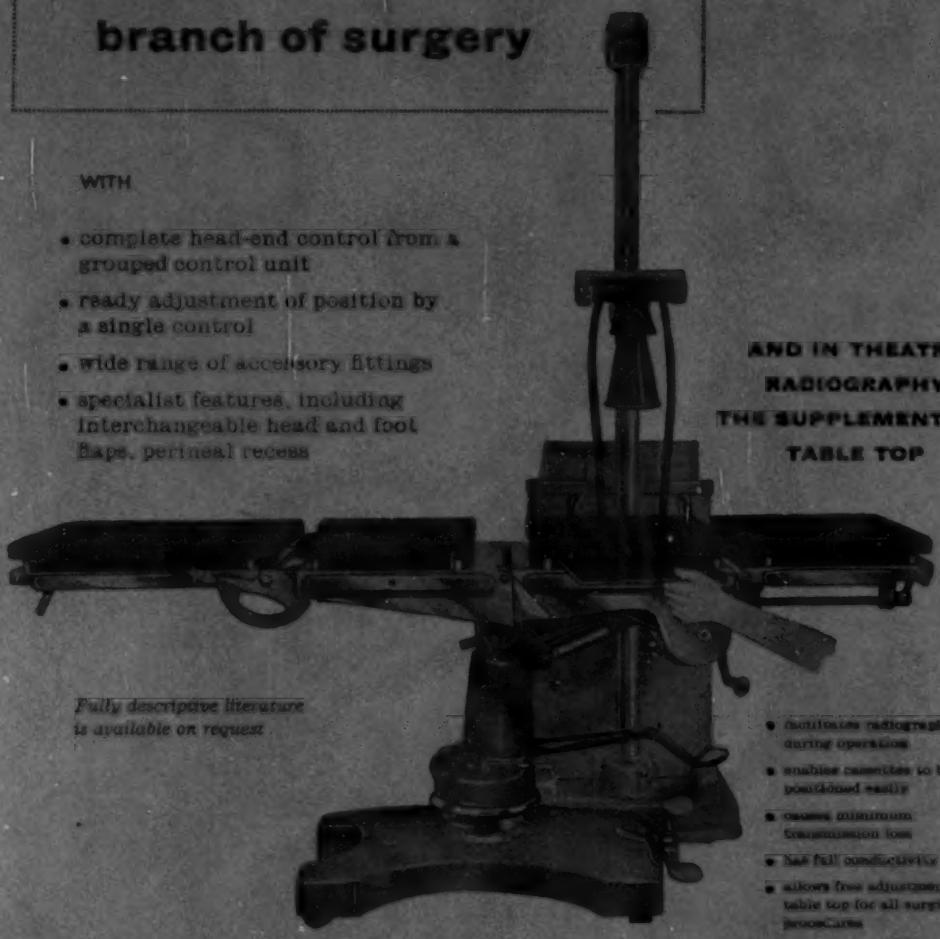

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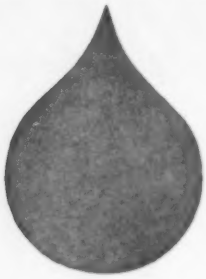

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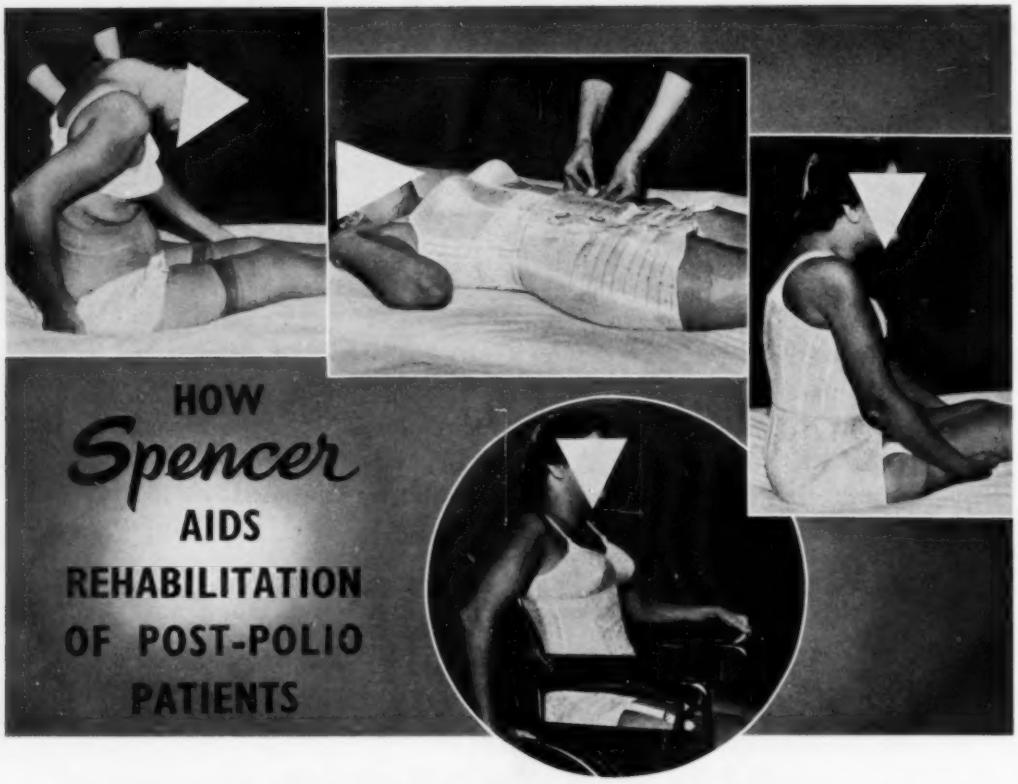

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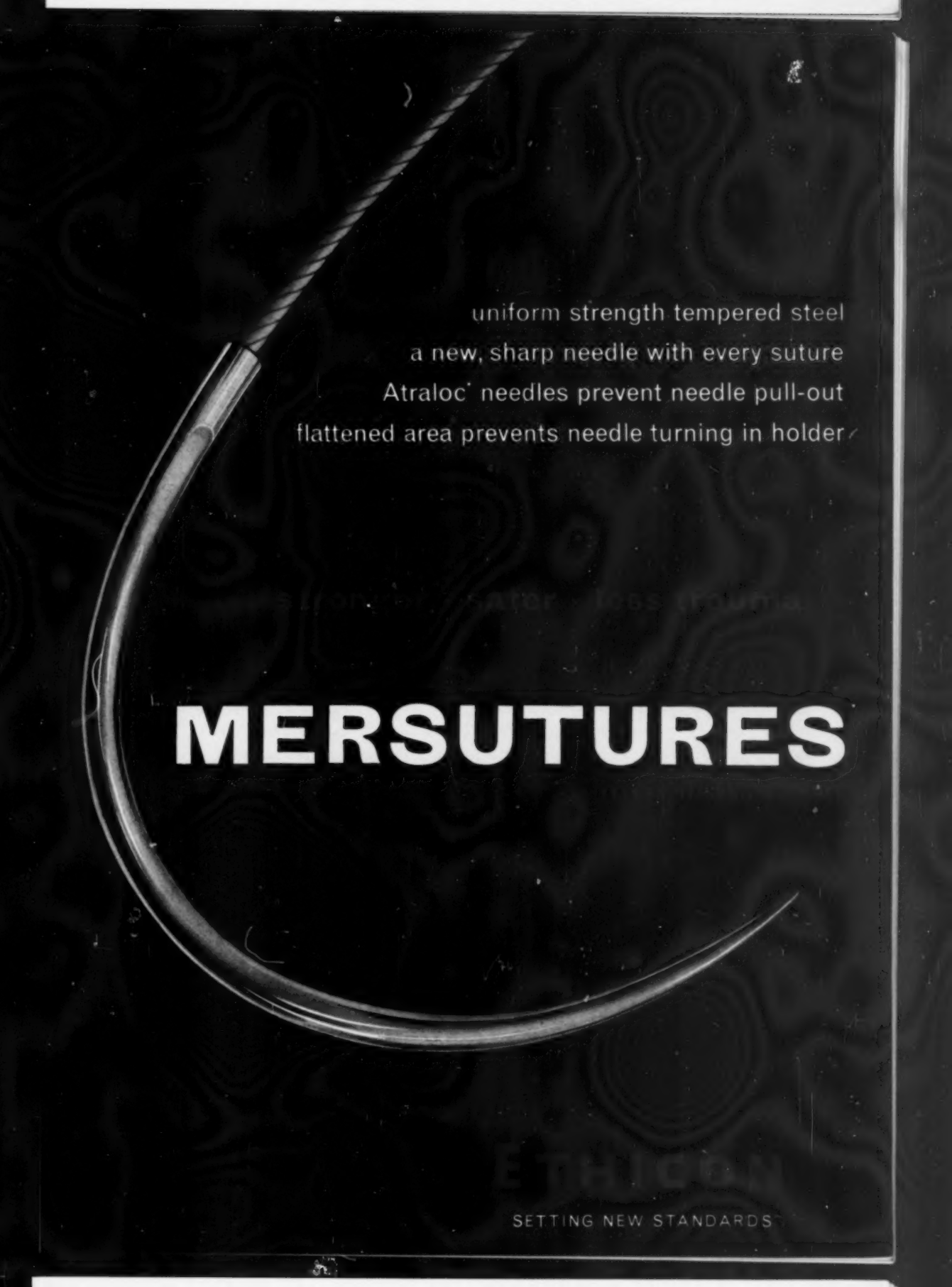
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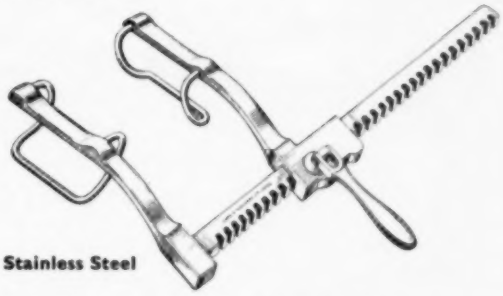

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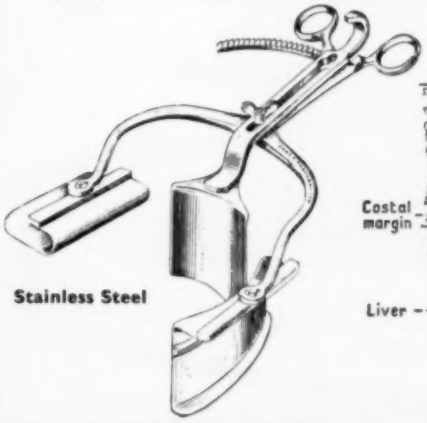

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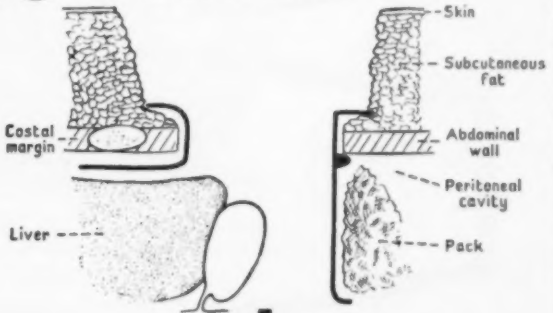

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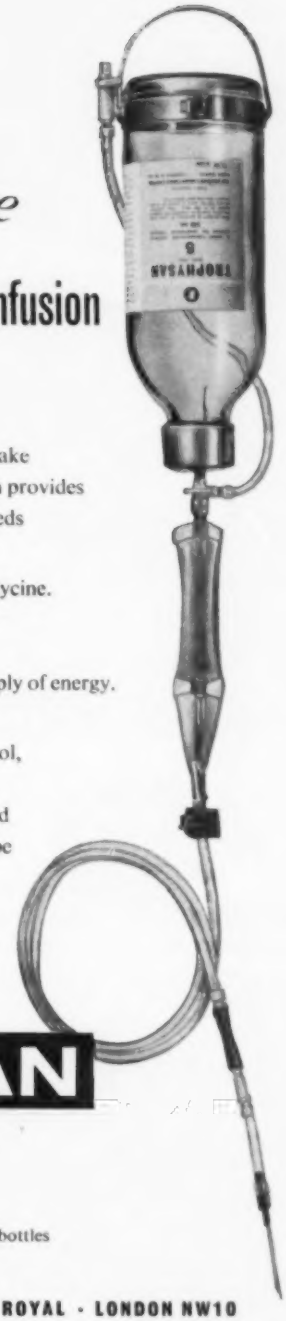

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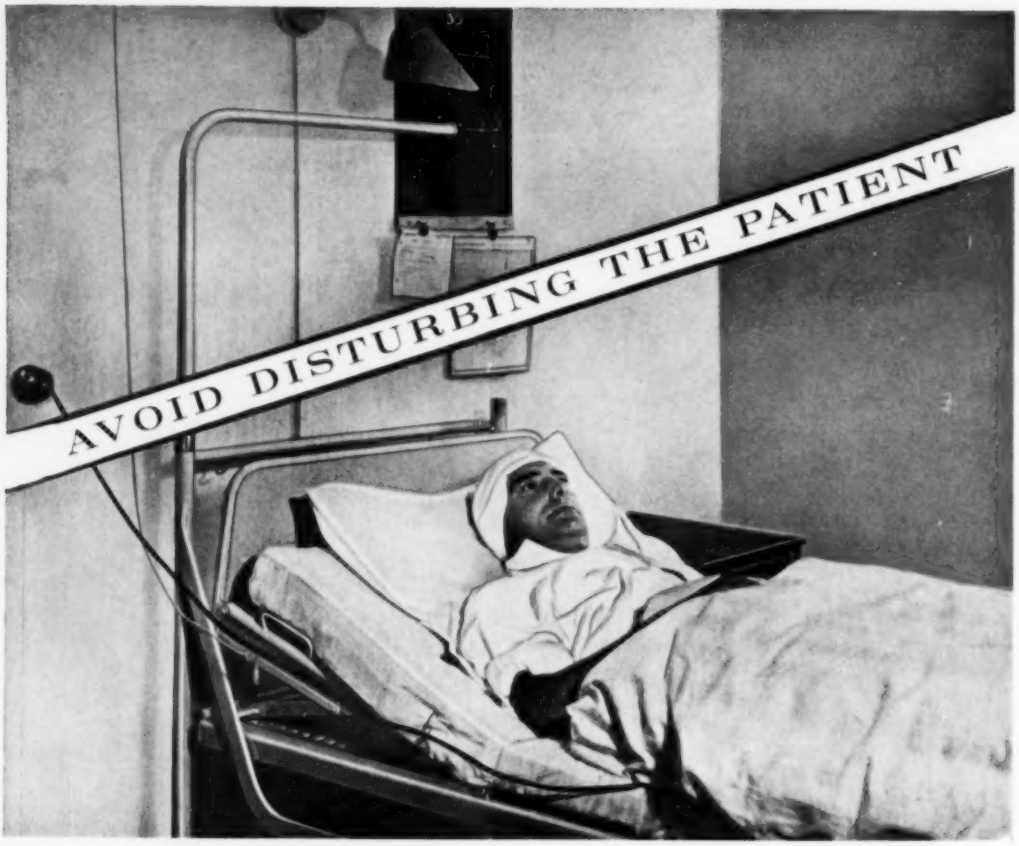

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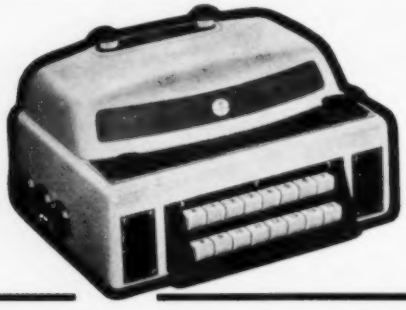

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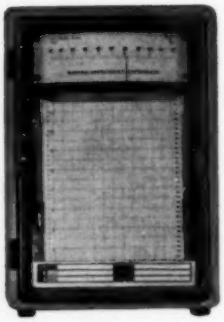

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THE
BRITISH JOURNAL OF SURGERY

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THE COMMON BILE-DUCT GROOVE IN THE PANCREAS

BY W. J. LYTLE
ROYAL INFIRMARY, SHEFFIELD

THE pancreatic part of the common bile-duct lies hidden and remote. Behind the first part of the duodenum the duct sinks out of sight into the pancreas and its thin walls escape detection by the fingers. Here the bile-duct follows no set course. It may lie on the posterior surface of the head of the pancreas or be deeply embedded in its substance; it may pass down close to the left border of the second part of the duodenum or lie as far away as 2 cm. from the duodenal wall.

FIG. 222.—Palpation of the bile-duct groove.

Whether by design or chance, it is fortunate that wherever the bile-duct may lie, it usually reveals its position by the groove which it makes on the back of the pancreas.

PALPATION OF THE GROOVE

Some years ago I came upon the common bile-duct groove in the pancreas and found a simple way of palpating it.

With the fingers of the left hand passed behind the second part of the duodenum, and the thumb in front of the head of the pancreas to act as a counter pressure, the common bile-duct groove can be readily felt (*Fig.* 222).

It is not necessary to mobilize the duodenum, for the fingers slip easily behind its second part to reach the groove which passes down on the back of the pancreas near the left margin of the descending part of the duodenum.

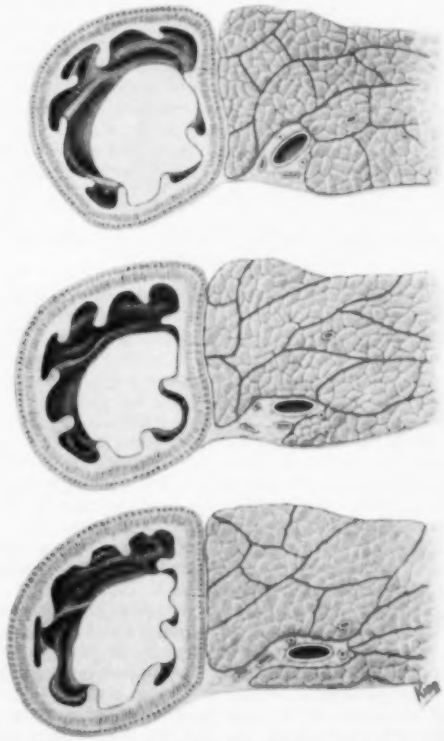

FIG. 223.—Transverse sections of the second part of the duodenum and head of the pancreas viewed from the caudal aspect, showing the varying positions of the bile-duct in the pancreas.

The groove consists of a hollow or furrow on the back of the pancreatic head, with raised margins of pancreatic tissue on each side. The pancreas has the consistency of soft rubber and this allows the groove to be easily felt.

The fingers cannot, of course, feel the thin-walled duct, but the hollow or recess where the duct lies gives to them an impression of the duct itself.

When the duct lies deeply the groove can still be identified. Transverse sections of the second part of the duodenum and the head of the pancreas (*Fig.* 223) show that when the bile-duct lies submerged it occupies an inlet in the pancreas. Not only is this so, but the entrance to the inlet in the pancreatic tissue lies open and the exit from the duct is directed posteriorly and to the right. The fingers of the left hand, sliding behind the descending part of the duodenum, can insert themselves between the margins of the pancreas on each side of the inlet and thus reach the deeply lying duct.

With a knowledge of the groove the operator can palpate it to detect stones lying in the lower portion of the common bile-duct, and be more certain that no stone is left behind when he finds the groove empty. The groove, as will be mentioned later, is of use to the surgeon in other ways.

Difficulty is experienced at first in finding the groove, but a knowledge of its outline and position can be gained by passing a bougie down the duct. The groove can then be more easily recognized as the instrument is being withdrawn. With practice the groove is readily felt in most cases and when its value is recognized it will be examined as a routine in operations on the biliary tract. In a recent series of 200 operations for gall-stones, where a record was kept, the groove was felt in 186 cases, giving a failure rate of 7 per cent.

THE ANATOMY OF THE BILE-DUCT AND ITS GROOVE

The pancreatic part of the common bile-duct extends from the cephalic end of the head of the pancreas to where the duct enters the wall of the second part of the duodenum. The duct in its groove usually presents a gentle curve as it passes downward and to the right, but it may also be straight or at times sharply curved. The lumen both above and in the pancreas normally measures from 0·5 to 1 cm. transversely and is uniform until it suddenly narrows as the duct enters the duodenal wall. Its length in the pancreas varies from 3 to 5 cm. and the duct usually passes down close to, or occasionally in direct contact with, the left border of the second part of the duodenum, but as already stated may be as far away as 2 cm. from the duodenal wall.

The bile-duct has a very generous blood-supply which in the pancreatic part comes mainly from the retroduodenal artery (posterior superior pancreaticoduodenal artery). This vessel commonly arises from the gastroduodenal artery and crosses from left to right in front of the bile-duct, usually just above where the duct enters the groove. The artery and its branches pass down in the groove with the bile-duct and the accompanying veins, lymphatics, and nerves.

Many have investigated the anatomy of the bile-duct in the pancreas by dissection, but my own studies were made from transverse sections of the duodenum and head of the pancreas which show clearly the varying position and contour of the space in the pancreas where the duct lies.

Dissections of the Bile-duct.—Hollinshead (1957) reviews the anatomy of the lower part of the common bile-duct and quotes amongst others the work of Baldwin (1911) and Smanio (1954), whose findings are very similar. Baldwin in his classic

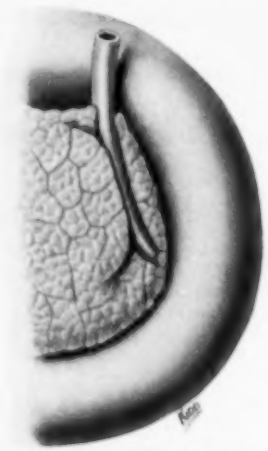

FIG. 224.—Posterior aspect of the duodenum and head of the pancreas showing a pancreatic lingula overlapping the bile-duct. (*After Smanio.*)

paper dissected 100 bile-ducts in the pancreas but Smanio worked on 200 specimens of the head of the pancreas and duodenum. These two observers found that the bile-duct lay in a groove in the pancreas, completely uncovered throughout its length and breadth in 15 per cent of cases (Baldwin) and 16·5 per cent (Smanio). In 85 per cent (Baldwin) and 83 per cent (Smanio) some portion of the duct, and at times its whole length, was partially or completely covered by pancreatic tissue. In one of Smanio's cases the bile-duct lay in front of the pancreas, a rare anomaly which has also been noted by other observers.

Smanio found that where the duct lay deeply it was overlapped by a lingula or tongue-shaped process of pancreas, arising usually from the pancreatic head on the left or caudal side of the bile-duct (*Fig.* 224). This lingula was either long or short according to the length of duct covered; broad or narrow, varying with the breadth of duct which was overlapped, and thin or thick, corresponding to the depth at which the bile-duct lay in the pancreas. In these 83 per cent of cases, no matter how deeply the duct lay, or how extensively it was covered by pancreatic tissue, Smanio was able in all of them to find a plane of cleavage down to it between the overlapping lingula and the back of the head of the pancreas. It is through this plane of cleavage that the fingers are able to reach a deeply lying duct. In 3 cases it was necessary to divide a small extension of the pancreatic tissue so as to free the duct completely.

Transverse Sections of the Duodenum and Pancreas.—One hundred transverse sections of 32 specimens of the second part of the duodenum and head of the pancreas were examined and sketched

(*see Fig.* 223). In 17 sections the common bile-duct was completely uncovered in its groove, in 83 the duct lay deeper in the pancreas. In 77 of the latter the duct was overlapped by a leaf or process of pancreas arising from the head on the left side of the duct, with a cleft in the pancreas leading outwards from the duct. This process of pancreas which corresponds to the lingula, described by Smanio, was either thick or thin and partly or completely covered the duct. In 6 sections the bile-duct was enclosed in the pancreas with no cleft to the surface.

Summary.—It is clear from these dissections and transverse sections that there is no normal arrangement of the duct in the pancreas, but the following points emerge:—

In roughly one-sixth of the dissections of Baldwin and Smanio, and also in the transverse sections, the duct was completely uncovered in its groove.

In the other five-sixths of the transverse sections, and also in the dissections by Smanio, the bile-duct was partially or completely covered by pancreas. In all but a few of these the duct was overlapped by a process of pancreas leaving a cleft in the pancreatic tissue which led backwards and to the right from the submerged duct to the posterior surface of the pancreas.

The groove should be easily felt in the uncovered duct. In the deeply lying duct, the fingers of the left hand, as they pass under the second part of the duodenum, readily enter the cleft to feel the space where the duct lies. As the fingers slip into the cleft the covering flap of pancreas is often felt as it is raised off the duct.

THE GROOVE IN SURGERY

Surgeons must have often felt the common bile-duct groove in the pancreas without realizing that it not only reveals the position of the pancreatic part of the duct, but gives some idea of its shape and size. The groove is not easy to identify at first, and it takes some time to appreciate it thoroughly and to realize its surgical value.

Stones in the Common Bile-duct.—The operator is enabled, by a knowledge of the groove, to ascertain the exact position of the bile-duct in the pancreas and to detect stones lying there; to remove them more easily, and to make more certain that no stone is left behind when he feels that the groove is empty. A bougie passed down the duct is felt to lie in the groove and helps the beginner to identify it. The passage of the instrument into the duodenum is confirmed by feeling the collar-like sphincter of Oddi around it at the lower end of the groove. When the pancreas is enlarged and firm the groove is more easily felt.

Obstructive Jaundice.—When the groove has become familiar to the surgeon it helps to distinguish between cases of stone in the common bile-duct, cancer of the head of the pancreas, cancer of the pancreatic part of the bile-duct, cancer of the papilla of Vater, and chronic pancreatitis giving rise to obstructive jaundice. The fingers can often determine the exact position of the stone or tumour mass in relation to the groove, and thus determine with more certainty the site and cause of the obstructive jaundice.

Repair of Bile-duct Injuries.—It is probable that end-to-end anastomosis of the bile-duct, which preserves the sphincter of Oddi, is to be preferred to any other method of surgical relief for a damaged duct. In any type of repair the upper segment of the damaged duct must be found, however difficult this may be, and here its blood-supply is usually good. To obtain good union, the ends of the duct when

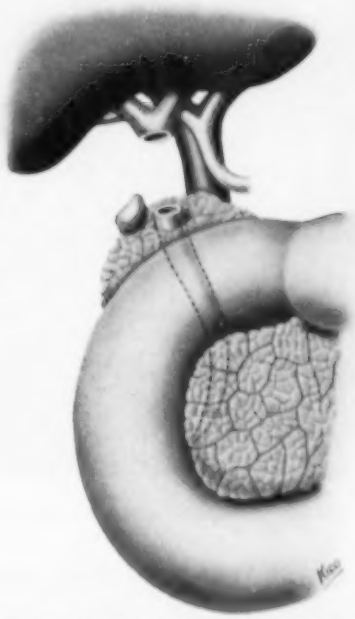

FIG. 225.—The ends of the damaged bile-duct are shown ready for suture. The blind end of the lower segment of the bile-duct is found by following up the groove, shown in dotted lines, and by the proximity of the common bile-duct lymph-node.

cut and ready for suture should bleed freely and be healthy and free from fibrosis.

By using the bile-duct groove it is possible to come upon the closed end of the lower segment of the duct more easily without damaging its blood-supply. This, I believe, will lead to a change in the surgical technique of repair and to the less frequent use of biliary-intestinal anastomosis.

The usual method of finding the closed end of the lower segment of the duct is by dissecting and freeing the duct from the back of the pancreas by the Lahey and Pyrtek (1950) technique. This is a sure way of securing the lower end, but the operation is difficult and tedious owing to bleeding from pancreatic vessels. The dissection also damages the blood-supply of the pancreatic part of the duct and releases pancreatic ferments which may digest the duct and catgut stitches at the suture line. The Lahey operation therefore requires the use of silk sutures for repair and T tubes to ensure union and prevent narrowing at the anastomosis. These tubes may have to be left in position for prolonged periods; they

often become encrusted and plugged, thereby damaging the wall of the duct.

By following up the bile-duct groove in the pancreas with the fingers and thumb of the left hand, the blind end of the duct can usually be found, where it is buried in the upper end of the head of the pancreas, without damaging its blood-supply or the pancreatic tissue in which it lies (*Fig.* 225). The common bile-duct lymph-node, which frequently lies at the upper end of the groove, behind and to the right of where the blind extremity of the duct should lie, is often an additional aid. It should rarely be necessary to open the duodenum to pass a probe up to the blind end from below.

It is an invariable practice, and experience shows how important it is, to use a T tube to prevent a stricture developing at the suture line when the lower end of the bile-duct has been dissected out from the back of the pancreas. If the duct has been isolated at the upper portion of the head of the pancreas with intact blood-supply and undamaged pancreas, then an indwelling supporting drainage tube, with its many disadvantages, appears to be unnecessary. It is advisable instead to place drains of corrugated rubber and perforated tubes on each side of the anastomosis to prevent bile collecting locally and leaking into the peritoneal cavity.

Where there is a stricture and the continuity of the bile-duct remains, the groove is also useful for locating the site of the stenosis amid the dense fibrous tissue.

Madden (1958) has operated on 4 cases since 1950 by end-to-end suture, using fine silk without the use of an internal splint or T tube, and these patients have remained well for 7 years, 55 months, 30 months, and 20 months respectively. Since 1954 I have repaired duct injuries, some with extensive damage, in 6 patients (7 operations) using 5 to 7 fine chromic catgut stitches on an atraumatic needle for end-to-end suture and without the use of a T tube or internal splint. While all these patients remain well, in only the first three, now 5, 3½, and 2 years since their operations, can it be claimed that the good results are likely to be permanent.

Injured ducts are rarely seen in this country, and although the numbers presented are few and the period since operation short, the results suggest that, where a technique is used to preserve a good blood-supply to the segments of the bile-duct, satisfactory union without subsequent stenosis is possible by end-to-end suture, without the use of a T tube or internal splint.

SUMMARY

The common bile-duct groove on the back of the head of the pancreas and the method of palpating it are described, together with some of its uses in the surgery of the biliary tract.

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GAS CYSTS OF THE LARGE BOWEL SIMULATING MULTIPLE POLYPOSIS

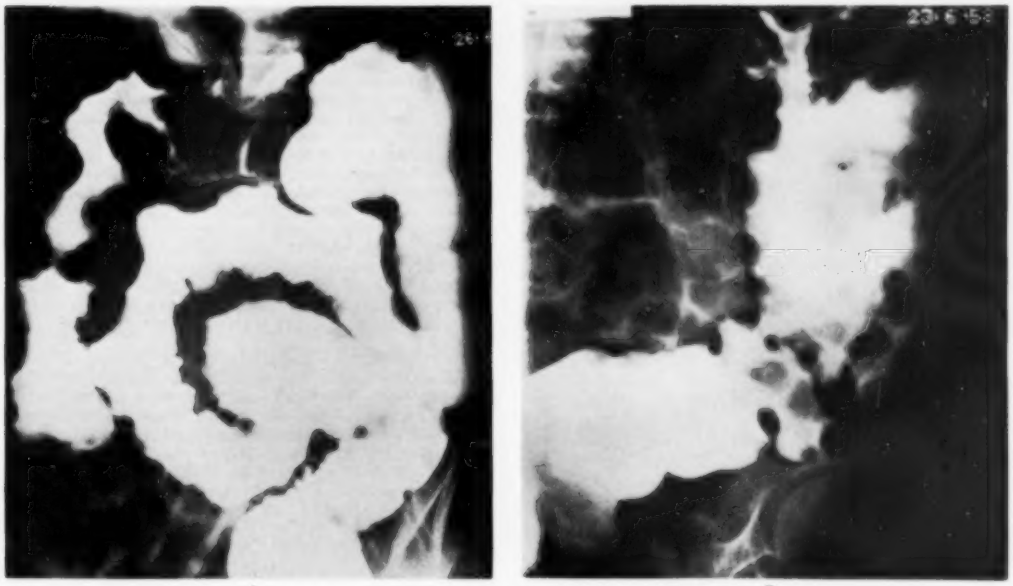
BY R. Y. CALNE, LONDON

GAS cysts of the intestines—pneumatosis cystoides intestinalis—were first described in man in 1730 by Du Vernoi. They are rare in man, some 300 cases having been reported, but they are commonly found in animals, especially in the pig (Hunter, 1837). Koss (1952) described a case and reviewed the world's literature of 255 cases. During the last few years increasing numbers of cases have been described in the English and American literature (Sherwin and Messe, 1952; Thoma and Hennigar, 1952; Briggs, Sifre, and Overholt, 1954; Kreeger and Littman, 1954; Massachusetts General Hospital Case Records, 1954, 1958; Matthews, 1954; Anton, Burger, Hoffman, and Weiner, 1955; Griffiths and Nash, 1955; Roundthwaite, Palmer, and Gardner, 1955; Witkowski, Pontius, and Anderson, 1955; Benson and Raitt, 1956; McGee, Penny, and Williamson, 1956; Marshak, Blum, and Eliasoph, 1956; Pemberton, Smith, and Holman, 1957; Edwards, 1958; McGregor and McKinnon, 1958; Yunick and Fradkin, 1958).

Gas cysts, which are commoner in men than in women (Nitch, 1924), are found anywhere throughout the small and large bowel, and the areas involved may be localized or very extensive. In man they occur from infancy to old age, but they are seen mostly during middle age. About half the cases are associated with gastro-intestinal disease, in particular stenotic duodenal ulceration, in which cases the upper jejunum usually contains most of the gas cysts (Koss, 1952). Gas analysis of the cysts reveals about 90 per cent nitrogen, 7·5 per cent carbon dioxide, and 2·5 per cent oxygen (Mujahed and Evans, 1958). This is in agreement with the first analysis of gas in pneumatosis by Cavendish, as quoted by Hunter (1837) from his specimen of hog intestine: "it contained a little fixed air (carbon dioxide) and the remainder not at all inflammable, and almost completely phlogisticated (nitrogen)".

Gas cysts may be found incidentally at post-mortem examination, operation, or upon X-ray examination. Cysts present at operation have been found to have disappeared on subsequent examination (Jackson, 1940). In those cases presenting with symptoms the most common is diarrhœa, although occasionally the cysts may cause pneumoperitoneum, volvulus, intestinal obstruction, or intussusception (Koss, 1952), and there is one fatal case reported of spontaneous rupture of the colon (Rosenbaum, 1957).

In the large bowel diagnosis rests on barium X-ray studies and sigmoidoscopic appearances. In both of these the similarity to polyposis has led to error in diagnosis (Alstraedt and Fielder, 1954; McKay, 1955; Ramos and Powers, 1957). The following case demonstrates this similarity. In view of the symptoms in this patient and the possible sequelæ of a watching policy, a distinction is perhaps academic, the desirable treatment of resection being common to both gas cysts and multiple polyposis.



A B

FIG. 226.—Barium enema showing multiple round translucent filling defects throughout the colon. A, Full plate; B, Detail study.

A B

FIG 227.—Photograph of colon from in front. A, Intact; B, Opened to display 'cobble stone' appearance of mucosa.

CASE REPORT

A housewife aged 45 years was admitted to the Royal Free Hospital (under the care of Sir Daniel Davies) on Aug. 25, 1958, with a complaint of constipation for two years. She had been in hospital several times in the last six years for treatment of bronchial asthma, which required ACTH therapy. A bilateral Caldwell Luc was performed in 1957 for chronic sinusitis. In 1956 she had a subtotal hysterectomy for fibroids and during the operation it was noticed that the colon was distended and a flatus tube was passed with little effect. Since this operation she had become increasingly constipated. She often felt that she had a full rectum, but could only pass a little mucus. Defæcation was sometimes accompanied by a central abdominal griping pain. She had passed a little bright red blood three months before admission. Her appetite was good. She had not vomited. She had lost 7 lb. in weight in the past year, her weight on admission being 112 lb.

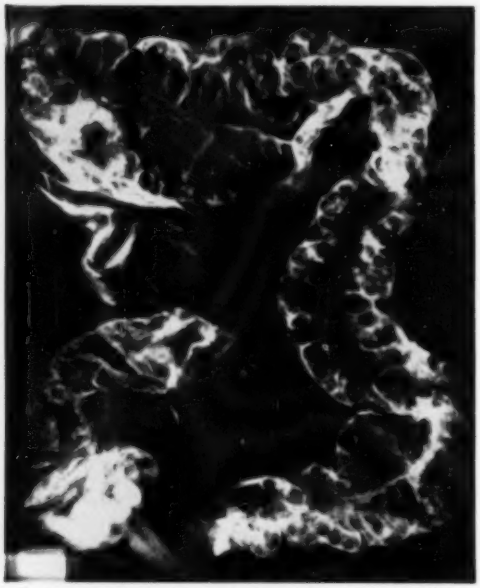


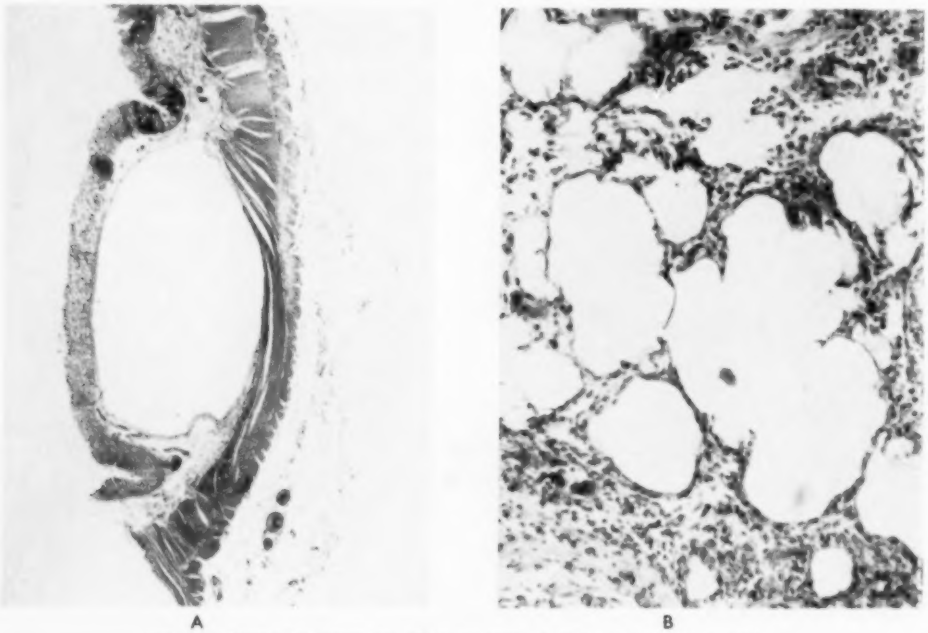
FIG. 228.—Radiograph of excised specimen showing multiple gas cysts.

ON EXAMINATION.—She was a rather introverted lady of fairly healthy general appearance. Her abdomen was moderately distended and hyperresonant into percussion. No other significant abnormality was found.

Sigmoidoscopy revealed a smooth round polypoid lesion at 25 cm. Barium enema showed large redundant loops of pelvic and transverse colon with multiple round filling defects throughout the colon, which could have represented polyps, though in retrospect the lesions had wide bases and an excessive translucency (*Fig.* 226).

AT OPERATION.—Laparotomy was performed (Mr. Qvist) and the colon was found to be dilated with large redundant loops of pelvic and transverse portions. On palpation the whole of the large intestine from cæcum to lower pelvic colon contained numerous polypoid lesions. There was no external evidence of malignancy, but multiple spherical clear cystic lesions varying in size from a pin-head to a pea were to be seen pedunculated from the serosal aspect of the large bowel. In view of a presumptive diagnosis of multiple polyposis, a total colectomy was performed with a one-stage ileorectal anastomosis.

The patient made an uneventful recovery and was discharged three weeks after operation.



A B

FIG. 229.—Histological appearance of the gas cysts. H. and E. (A, × 6; B, × 150.)

MACROSCOPIC EXAMINATION.—The specimen showed multiple gas cysts of the whole large intestine. These projected in a polypoid manner producing a 'cobble stone' appearance on the mucosal aspect (*Fig.* 227).

RADIOGRAPHY OF THE SPECIMEN.—This revealed numerous gas cysts with an extensive distribution (*Fig.* 228).

MICROSCOPY (*Fig.* 229).—The large submucous cysts had fibrous walls and an incomplete lining of flattened epithelium. The overlying mucosa and underlying muscle-coats were thinned. Many of the small cysts had no definite lining, others were lined with irregular syncytial-like masses of cells. The septa between groups of small cysts were breaking down in places and the cysts were coalescing. The intervening fibrotic submucosa was heavily infiltrated by lymphocytes, plasma cells, giant cells, and especially eosinophils. Similar cells were present in the mucosa (Dr. I. Filshie).

DISCUSSION

The aetiology of gas cysts has been much debated and as in all fields of medicine where ignorance is nearly complete, the number of theories put forward is considerable. Thus a neoplastic origin, the result of bacterial gas-producing organisms, mechanical pressure changes, dietary deficiency, and changes in *p*H resulting in gas production, have all been suggested as causative agents (Koss, 1952). However, there appears to be no direct evidence in favour of any of these.

Chronic asthma, ACTH therapy, and eosinophilic infiltration around the cysts in this case are perhaps of interest, especially as two similar cases of chronic asthmatics with pneumatosis have recently been described. Both were middle-aged females and in both the disease was limited to the large bowel (Matthews, 1954; Creevey, Cohen, and Riley, 1958).

SUMMARY

A case of extensive gas cysts of the colon presenting as constipation, simulating multiple polyposis, and treated by resection is described.

Acknowledgements.—I wish to thank Mr. Qvist for his help and criticism and for his and Sir Daniel Davies's permission to publish this case. I am indebted to Dr. Filshie for the histological report; to Mr. Lefanu for translating Du Vernoi's article; to Mrs. Blake for help with the bibliography, and to Mr. Phillips for the illustrations.

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THE PROBLEM OF DISTENSION IN LOW SMALL INTESTINAL OBSTRUCTION

WITH SPECIAL REFERENCE TO ITS MANAGEMENT AT OPERATION

By S. R. SHAR, Birmingham

In the past few decades the prognosis of acute small intestinal obstruction has brightened considerably, but even to-day the mortality and post-operative morbidity rates remain disturbingly high.

Since McKittrick and Sarris (1940) urged early operation, the results of treatment have shown a distinct improvement. No doubt this is mainly because many cases of strangulation have received timely attention, whereas a policy of temporization might have led to disaster, when a pre-operative diagnosis of simple obstruction had been entertained. Nevertheless, strangulation is still the major single cause of death in any substantial series.

A more complete understanding of fluid and electrolyte balance has dramatically improved the outlook in obstructions, especially when the level is high in the small intestine. In restoring the physiological balance of a depleted patient with a non-strangulating obstruction, four to six hours spent in the pre-operative phase can be of inestimable value. The patient is then far better equipped to tolerate anæsthesia and an operation which may be lengthy.

LOW SMALL INTESTINAL OBSTRUCTION

When the site of obstruction is beyond the jejunum, particularly in the distal ileum, the pathogenesis of the condition is far more insidious. In high small-bowel occlusion, little absorption takes place and vomiting is an early feature, consequently attracting attention in the initial stages. On the other hand, the length of intestine proximal to obstructions lower down is sufficient to allow some absorption, while the dilated lumen of several feet of ileum can accommodate a large volume of fluid without regurgitation.

At first these obstructions are often incomplete, and it is not uncommon for patients to procrastinate in the hope that their colicky pains are due to a digestive upset and will soon settle. Unlike high obstructions where fluid loss is rapid and this is paralleled by a decline in general condition, there is progressive distension of the proximal intestine, which is sometimes slow to manifest itself. Frequently, these patients first seek medical advice when the condition is relatively advanced and the abdomen is already grossly distended. The onset of vomiting in occlusions at low levels is a sign that several days have elapsed since the inception of the obstruction. In its development, therefore, a low obstruction differs fundamentally from one near the duodeno-jejunal flexure; the superadded problem of distension is now the factor of overriding importance.

THE EFFECTS OF DISTENSION

Even in this enlightened age, the baneful effects of distension are responsible *per se* for more morbidity and mortality in simple non-strangulating obstructions than the sum total of all other deleterious components of the syndrome. The intestine immediately above the site of occlusion contracts vigorously in an effort to propel its contents distally. This attempt being thwarted, the bowel lumen dilates to accommodate the contents which are soon augmented by the advancing fluid and gas from the upper reaches of the intestine. The accumulation of intestinal contents produces a rise in the intraluminal pressure. This at first stimulates absorption (Elman and Aird, 1935), but soon the optimal pressure for the outward passage of fluid is exceeded, and a further rise leads to compression of the veins draining the mucosa. As a consequence there are congestion and œdema of the bowel wall, while the rate of secretion into the lumen is sharply increased, causing a further rise in intraluminal pressure and so a vicious circle is set in motion (van Zwalenburg, 1932).

With the progressive distension of the intestine the musculature becomes anoxic and fatigued and its contractility diminishes, while compression of the wall impairs its viability. Resultant upon the latter the gut may perforate along its antimesenteric border, or develop mucosal ulcers which theoretically facilitate absorption of toxic products from the lumen if the obstruction is relieved and the circulation improves. When the obstruction has reached the above stage the condition of the patient is grave, and results of treatment leave much to be desired.

The cause of collapse and death at this juncture is controversial; it may be reflex, from afferent stimuli arising in the mesentery of the distended gut; there may be transperitoneal migration of 'toxins' through the injured wall whose permeability is increased; or, finally, there may be frank peritonitis, the lethal fury of the organisms multiplying in the gut being unleashed in the general peritoneal cavity.

It should be reiterated that, together with unrecognized strangulation, progressive distension is the chief agent which causes the still alarmingly high mortality-rate in intestinal obstruction. Obviously, early diagnosis followed by rapid relief of the obstruction is the ideal method of preventing such a state of affairs from developing. However, from the very nature of the pathological conditions causing a low small intestinal block, the obstruction is at first partial, e.g., gall-stone obturation and ileo-cæcal carcinoma; distension is frequently well advanced when the clinician is first confronted by such a patient.

GASTRO-INTESTINAL INTUBATION

At the end of the last century, Turck employed a sprung rubber tube whereby he obtained samples of the duodenal juice. Since his pronouncement, dozens of variants have been described in the literature. The forerunners of present-day gastroduodenal tubes were those of Einhorn and Gross, devised separately in 1910. These were single-lumen lengths of rubber tubing with terminal metal tips meant to be introduced nasally or orally and passed down the

œsophagus into the stomach, whence they were guided through the pylorus.

When, in 1931, Wangensteen applied gastro-duodenal intubation to the treatment of intestinal obstruction a great milestone was reached. What could be more ideal and, ostensibly, more simple than to decompress the distended gut by applying suction to an indwelling tube? Wangensteen and Paine modified the Levine tube by extending the perforations several inches proximally so that suction could be applied simultaneously in the stomach and duodenum.

It was soon apparent that gastroduodenal suction was not entirely satisfactory in decompressing the whole length of the small intestine. The obstructed intestine is distended by fluid as well as gas, and laden segments of bowel which are kinked by their own weight prevent the equal distribution of hydrostatic pressure, as would be the case if the intestines were a continuous column of gas alone. Therefore, the short tubes rely for their action on the reflux of the contents of a distal loop into the empty one above.

For this reason the long intestinal tube was developed, the aim being to pass the tube down to the level of the obstruction, deflating the whole gut proximally. Nearly a quarter of a century ago the double-lumen tube was introduced (Miller and Abbott, 1934), and this was soon followed by those of Harris, Johnston, and Cantor. All these tubes rely on a balloon at or near the end for propulsion along the bowel. The balloon is either weighted with mercury or inflated with air after it has entered the duodenum. A high percentage of satisfactory intubations is claimed by those who advocate the use of these tubes and a variety of accessory aids has been employed to negotiate the pylorus, such as fluoroscopy, posturing, wire stylets, metal weights, and powerful magnets.

However, the successful execution of this manœuvre is by no means universal. Under normal conditions it is relatively simple to coax a tube into the duodenum, but in obstruction an entirely different set of conditions is encountered. Aird (1950) states that in addition to an alteration in the pyloro-duodenal angle, the hydrostatic pressure increases proximo-distally in the distended intestine. Thus an advancing tube must pass against the stream of intestinal content from a low pressure to a higher one. A loop emptied by suction is immediately filled again by a rush of fluid from below. On occasion, even when the tip of the tube has advanced beyond the pylorus, the surgeon has the mortifying experience of witnessing it curl back into the stomach.

Certainly, the more practised the intubator, the higher is his success rate likely to be, but most surgeons will admit that they fail to pass the tube beyond the pylorus in a significant percentage of cases.

At least 80 per cent of intestinal gas is swallowed air, and Paine (1954) stresses the importance of having an indwelling tube whose perforations exert suction in the stomach in order to keep that organ empty. None of the long tubes has perforations extending along the major part of the length. Hence he advocates the introduction of a second tube into the stomach, even when the long tube has reached the level of the obstruction.

In spite of the difficulties attending intestinal intubation in obstruction, conservative decompression completely revolutionized the prognosis and therapy of small intestinal obstruction. So successful was this manœuvre that surgeons relied increasingly on its use. In several centres there was a marked swing away from early operation while mortality figures improved. Carried away by their initial enthusiasm, many surgeons were tempted to persist with this therapy beyond the limits of its beneficial application, with the result that a number of lives were lost by delaying operation.

Second thoughts on this matter have served to place the use of gastro-intestinal suction in a more logical perspective. Although a mechanical obstruction may be simple rather than strangulating, and its relief by conservative means is initially successful, the nature of the occlusion is rarely known with certainty. Thus the surgeon may be lulled into a false sense of security. The following case illustrates this pitfall:—

A female, aged 72 years, presented for the first time with a history of intermittent colicky lower abdominal pains. Her bowels had been constipated for three days and she had passed no flatus for forty-eight hours. She had not vomited, although she had experienced nausea. A perforated appendix had been removed thirty years previously, and she underwent a hysterectomy for menorrhagia due to fibroids five years later. There were no symptoms pointing to biliary tract disease.

On examination the abdomen was grossly distended and the patient obviously had a low small bowel obstruction, although her general condition was fairly good. No abdominal tenderness was elicited on palpation. Plain radiographs revealed numerous fluid levels and coils of distended small gut, but no gas was visualized in the colon. The presumptive diagnosis was an adhesive obstruction, and it was decided to begin a trial of conservative decompression. A double-lumen Miller–Abbott tube was successfully passed, reaching the lower ileum in just over twenty-four hours. Within thirty-six hours the patient's condition had improved considerably, the distension had subsided, both clinically and radiologically, and she had passed small amounts of flatus per rectum. On the following day her condition suddenly deteriorated, and signs of general peritonitis were noted. Laparotomy was performed, revealing a perforation in the terminal ileum due to pressure by a large gall-stone which had lodged in the lumen. Singularly few adhesions from previous intervention were present.

No better example than the above can be quoted to illustrate the fallacy of purely conservative management of low small-bowel obstruction the cause of which is unknown. In mechanical intestinal obstruction tube decompression is no substitute for operation except in the circumstances cited below. When the patient's general condition is good, a preliminary attempt to pass a long intestinal tube is justifiable. Nevertheless, this procedure may be very tedious at the best of times and the process is exhausting to patients who are very ill. For this reason many practitioners do not attempt to use the long tube in even early cases of low small intestinal obstruction.

The method is to be discouraged when at the outset the general condition of the patient is poor. It is a far sounder course of action to pass a tube to keep the stomach empty, to spend a few hours resuscitating the patient if necessary by means of intravenous therapy, and to operate early. In this

way the cause of the obstruction may be attacked directly, if feasible, while regrettable instances, such as that quoted above, will be avoided. Furthermore, such a policy being a definitive one, will prevent wavering, while conservative therapy is marginally effective and the patient's condition may be deteriorating unobtrusively in the face of maintained distension. There will be far less morbidity after routine operation on all cases of non-strangulating mechanical low small-bowel obstruction (with the two exceptions cited below) than will be occasioned by giving the majority of these cases an extended trial on conservative management, even though a number of 'unnecessary' operations may be performed.

Nevertheless, a trial with conservative management in mechanical low small-bowel obstruction has a very definite place, particularly where this condition follows in the first few weeks after an operation such as the removal of a gangrenous appendix, or in cases of recurrent adhesive obstructions. Of course, in cases of a dynamic ileus, intestinal intubation coupled with intravenous therapy should be standard practice.

It is far easier to prevent the onset of distension than to combat the problem in an established case. Therefore, after elective or emergency surgery on the gastro-intestinal tract, the routine employment of nasogastric suction, until normal peristalsis is resumed, will maintain the stomach in a collapsed state, removing particularly any swallowed air and precluding distension.

In addition, the application of suction to a tube resting in the stomach during the anæsthetic induction will prevent reflux of intestinal contents into the air passages.

More recently, Smith (1952), of Minnesota, has developed a method of guiding a long tube into the intestines using a special technique involving a flexible stylet. He claims almost uniform success in a short time by this means. His tube naturally has great advantages where this procedure is indicated.

DISTENSION AT OPERATION

Every surgeon who is constantly meeting abdominal emergencies has been faced frequently at operation with the case of low small-bowel obstruction, either simple or strangulating, where the intestine is grossly distended. The bowel is friable, heavy, soggy, and often purple rather than pink in colour (*Fig.* 230).

Manipulation in such cases is fraught with many hazards:—

1. Because of the distension, it is often difficult to visualize and deal with the site of obstruction.

2. The bowel is easily contused by any but the gentlest handling.

3. The very weight of the distended coils may cause alarming tears in the mesentery, which sometimes injure important blood-vessels. Indeed, a split may already have appeared in the serous coat of the gut itself.

4. Anastomoses may be necessary between segments of gut with a great disparity in circumference.

5. Even with the outstanding relaxation afforded by modern anæsthesia, suturing of an abdominal wall in the presence of grossly distended intestine may be wellnigh impossible without producing disconcerting tears in the peritoneum and posterior rectus sheath.

If the gut is not decompressed, the existing distension is added to the post-operative ileus, seriously embarrassing respiration by pressure on the diaphragm. A further possible remote danger is that the distended bowel produces a fibrin-laden exudate which may later give rise to intraperitoneal adhesions.

All the above dangers could be counteracted to a large extent, if, as a first stage, the distension could

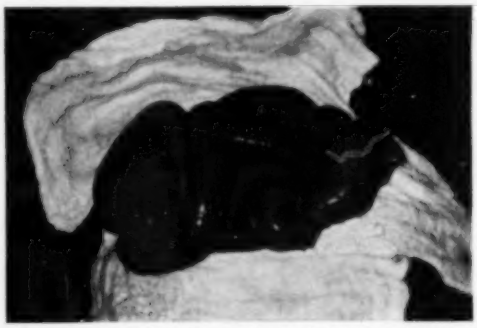

FIG. 230.—Gross distension of the small intestine which is frequently met in operations for small intestinal obstruction.

be reduced before an attempt was made to deal with the pathology. The oral passage of a tube by the anæsthetist, which is threaded along the intestine while suction is continuously applied, has been recommended by Smith (1956) and Alden (1956). Such a method, if skilfully performed, is certainly very useful, and has the additional advantage that a long tube can be left in situ post-operatively. However carefully this manœuvre is conducted, it will involve much manipulation of the intestines with the risk of trauma to the bowel wall. For most surgeons it is by no means easy to guide a tube from the stomach through the pylorus, along the duodenal loop, past the ligament of Treitz, and into the jejunum. Again, this latter procedure is often time-consuming, where saving of valuable minutes may determine the patient's fate.

In the introduction to his book, *Intestinal Obstructions*, Wangensteen (1955) states: "Distension, itself, constitutes a major surgical problem at operation frequently. Surgical techniques must be devised which will permit dealing adequately and safely with distension at operation. The patient whose distension remains unrelieved by attempts at accomplishing decompression by conservative means, constitutes a serious problem for the surgeon. The surgeon must learn to decompress the distended bowel at operation, in order that he may deal effectually, in certain instances, with the obstructing mechanism. When the surgeon masters the art of dealing with the item of distension at operation without visible traces of soiling, another important milepost will be reached in the reduction of the mortality of intestinal obstruction."

It is a simple matter to decompress the gut through a small enterotomy incision. Senn (1889) was among the first to recommend such a procedure.

Since that time the method has been used by many writers, but has never gained universal favour. The obvious danger is peritoneal soiling, which, though no longer as catastrophic as in the pre-antibiotic era, may easily tip the vital balance against the patient. Another reason for the lack of general acceptance of this method is that no uniformly successful instrument has been used to achieve satisfactory decompression.

A certain amount of experimental evidence has been produced demonstrating sudden collapse and death after rapid decompression of distended intestine. The most notable papers describing this phenomenon have been by Oschner and Storck (1936), Aird (1935), Elman (1934), Morton (1932), and Stone (1926). The thesis is that excessive handling of the bowel, especially in its vulnerable distended state, leads to mural damage. This allegation is directed chiefly against the stripping procedure where the distended gut near the duodenojejunal flexure is compressed between finger and thumb. Emptying is achieved by a distally directed milking action, either by drawing the bowel through the fingers, or by advancing the approximated digits towards the ileocæcal valve. Certainly, such a manœuvre can traumatize the intestinal walls, as a considerable amount of pressure may be required to maintain compression in order to prevent any of the intestinal contents from slipping into the proximal coils.

When the distension is relieved, the veins which may previously have been occluded are once more capable of draining the gut wall. In addition, mural damage having been sustained by the stripping or pre-existing distension, absorption of bowel contents by a previously less permeable mucosa is readily visualized. Potent vasodepressor materials may be swept into the general circulation, producing profound shock and even death. It is also conceivable that histamine-like agents, or other, as yet undefined, toxic substances may gain access to the blood-stream in this manner.

By the same token it is not difficult to explain the sudden collapse which may follow upon the untwisting of a gangrenous loop of colon in volvulus of the sigmoid. Here an important principle emerges. In a closed-loop obstruction, particularly where the venous drainage is obstructed, and where part of the intestine is no longer viable, the vessels supplying and draining the part should be ligated before attempting to relieve the obstruction. By so doing, many a mysterious death will be avoided.

Evidently there is a definite danger associated with the relief of distension, but this is no less when a simple obstruction is relieved and the gut contents are allowed to pass on naturally. It seems more logical to remove the contents of the dilated bowel, some of which may otherwise be absorbed through a damaged mucosa.

The crux of the matter is surely whether the distension has, in fact, damaged the bowel wall and rendered the mucosa more permeable, though not necessarily affecting the viability of that part of the gut. If this is the case, then decompression by emptying the bowel at operation can have no more dire effects than simple correction of the obstruction, provided that the act of operative decompression does not inflict further mural damage than is already present.

Superimposed on the presumably beneficial effect of removing the potentially harmful stagnant intestinal contents, decompression at operation has so many additional advantages that a successful expeditious method will save many lives.

A whole host of methods attempting to achieve decompression has been practised, ranging from the simple insertion of a wide-bore needle through a puncture wound in the wall of the intestine, to a variety of tubes (Moynihan, 1928; Holden, 1926; Cheever, 1932; Pool, 1913; etc.).

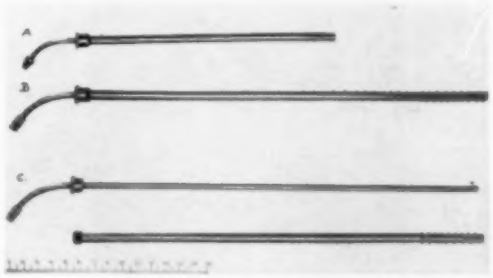

FIG. 231.—A, The standard Pool suction end, 7 in. long. B, The author's modification. C, The component parts—outer sheath and hollow shaft of the suction end.

Any such method is subject to four possible drawbacks:—

1. The danger of peritoneal contamination.
2. Damage to the bowel wall.
3. Incomplete emptying of the bowel.
4. The loss of a great deal of time.

Because of one or more of the above possibilities all methods previously described have failed to come into general use and have sunk into obscurity.

The author, bearing the four likely drawbacks in mind, has devised a technique which is as aseptic as any enterotomy can be, which is associated with a minimum of trauma, which empties the intestine completely, and which can be carried out fully in only a few minutes.

METHOD RECOMMENDED FOR DECOMPRESSION AT OPERATION

The only instrument needed is a modified Pool's sucker end (*Fig.* 231). (Specifications: length, 27·5 cm., and 2-mm. diameter perforations.) For descriptive purposes it will be assumed that the obstruction is located in the region of the ileocæcal valve.

After the peritoneal cavity has been opened a hand is inserted into the abdomen to ascertain the level of obstruction, i.e., the most distal loop of distended small intestine or the junction of dilated and collapsed bowel. The distended intestine is then exteriorized and covered with warm packs while the assistant supports the weight of the loaded loops.

A point is selected approximately 5 ft. (150 cm.) from the obstruction, and 4 seromuscular catgut Lembert sutures are inserted in the transverse axis of the intestine along its antimesenteric aspect. These sutures are not tied, but are left long and clipped individually. Two pairs of fine Allis forceps

are applied to the bowel wall between the two central sutures. An incision is made through the total thickness of the bowel wall just large enough to admit the point of the sucker, which is immediately inserted. The Allis forceps are removed to avoid trauma to the

FIG. 232.—The suction end is inserted into the lumen of the intestine which is collapsed on to the shaft, while the assistant holds the Lembert sutures aloft.

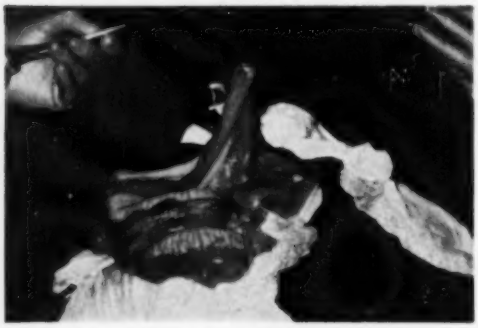

FIG. 234.—At the conclusion of operative decompression the intestine is now pink and collapsed.

gut wall while the assistant holds all four Lembert sutures aloft by grasping the clips (*Fig.* 232). This manœuvre produces an inverted V, the apex being the incision in the antimesenteric border of the intestine, while the two limbs are distended intestine. At this stage it is important to note that the surgeon has both hands free, one to advance the sucker and the other to manipulate the intestine.

By observing all the steps so far enumerated there is no danger of peritoneal soiling, as the non-gaseous contents of the intestine sink to the most dependent parts of each limb of the inverted V. The sucker is then guided into the proximal limb and gently advanced. At once the portion of the intestine which has been negotiated is seen to collapse on to the shaft of the sucker. Then, with the utmost care, the intestine is reefed along the entire length of the sucker, the surgeon making absolutely certain that the concertina-like corrugations are not too tightly squeezed together (*Fig.* 233). When the sucker is completely 'loaded' with intestine, at least

FIG. 233.—Several feet of small intestine have been reefed along the length of the sucker without squeezing the corrugations tightly together.

5 ft. of gut should have been completely decompressed. The portion proximal to this level may be emptied by careful stripping towards the sucker, or preferably through a separate incision if necessary.

It is often found that the proximal jejunum is so minimally distended that the latter is not required. The intestine is then allowed gently to slip off the shaft of the sucker while the instrument is withdrawn until its tip is just within the lumen of the gut, as was the case at the outset.

The direction of the sucker is now reversed and the procedure is repeated, emptying the 5 ft. of intestine proximal to the obstruction in a similar manner. The evacuation of not less than 10 ft. of intestine being achieved, the sucker is completely withdrawn and placed away from the operative field. The incision in the wall of the bowel is closed automatically by the tension exerted by the assistant on the Lembert sutures, which are then tied consecutively. To render the closure completely leak-proof the suture line may be buried by inserting a further seromuscular layer of continuous catgut. This method of closure is superior to the use of a purse-string suture which suggests itself as an alternative, as the lumen of the intestine is less encroached upon and the end-result is much more attractive æsthetically.

A great problem when attempting to decompress the intestine by enterotomy suction is the finding of a suitable instrument. Most sucker ends have a large terminal or side opening which soon becomes plugged by forcibly drawing the mucosa inwards. This undoubtedly damages the wall of the viscus and is undesirable, as are any sucker ends with

relatively few and large perforations, because not only do they damage the bowel, but they are inefficient.

The Pool type of suction end has an outer sheath with multiple very small perforations which is fitted over an inner hollow metal shaft. The suction exerted through each of the small perforations is not sufficient to damage the bowel by drawing in the mucosa, while the multiplicity of openings ensures effective suction without danger of plugging. Originally, the author used the standard Pool sucker of 7 in. length and it was soon apparent that this has two serious drawbacks, namely, that it was too short and that the perforations were too small. In long-standing obstructions there are numerous gritty particles in the lumen of the intestine as well as fluid and gas, and these soon block the standard-sized perforations of the Pool instrument, with the result that the sucker must be withdrawn and cleaned several times. Such an imperfect arrangement increases the risk of peritoneal soiling and consumes much time. Also, hardly more than 3 ft. 6 in. of œdematous intestine can be telescoped on to a 7-in. instrument without producing trauma. Accordingly, the author's modification was introduced, whereby the over-all length of the suction end is increased to 11 in. (a longer rigid tube becomes unwieldy), and the perforations have been increased in diameter to 2 mm., while their number is not reduced. With these dimensions the instrument proves ideal for the purpose. It is long enough to hold over 5 ft. of bowel, while the perforations are of sufficient calibre to remove solid particles from the lumen of the gut without becoming blocked, and at the same time they remain sufficiently small to preclude any risk of damage to the mucous membrane.

Using the technique described above and the instrument evolved, all the possible disadvantages of suction enterotomy are eliminated. There is no peritoneal soiling; emptying of the bowel is complete; no damage is inflicted on the mucosa; and the whole procedure can be conducted in no more than ten minutes.

Monks (1903) made some very simple but extremely useful observations on intestinal localization, and much of the modern surgeon's familiarity with the disposition of intestinal loops stems from his pioneer work. A fact to which he drew attention was that the intestine has a natural reefing action. This he demonstrated by threading the entire small gut on a 12-in. glass rod. This is confirmed *in vivo* by the fact that the tip of a Miller-Abbott tube no longer than 11 ft. can be made to appear at the anus while one end remains in the nose—this after traversing the entire length of the gastro-intestinal tract.

Moynihan (1928) was quick to see a practical application of this knowledge with regard to intestinal obstruction. Describing his method, he states that 8–10 ft. of bowel can easily be threaded on to a tube 6 in. long. This may be so with a normal intestine, but it is certainly impossible in the case of the heavy, friable, œdematous intestine which is distended by obstruction of long duration, without producing serious trauma. Moynihan's tube was a hollow glass cylinder (Bantock's tube) with a bevelled terminal opening. As stated earlier, this single wide opening is not by any means ideal.

Therefore, although operative decompression of the intestines has been practised for half a century, it has never gained general acceptance as a useful procedure because of possible peritoneal soiling and the lack of a suitable instrument, through the medium of which the entire manœuvre can be completed atraumatically, efficiently, aseptically, and in a very short space of time.

It has long been taught that exteriorization of loops of obstructed intestine induces severe shock and is decidedly harmful. However, to conduct intra-abdominal manipulations while distended coils impede access and are easily traumatized, is often far more deleterious to the patient. A great deal less harm will be produced if the intestine is exteriorized for the short time required to accomplish effective decompression, after which the gut is easily returned to the peritoneal cavity. The surgeon then has much more freedom of movement and can relieve the obstruction, free from the duress to which he is otherwise subjected. Vision is unimpaired, resections, anastomoses, or any other technical problems can be more easily dealt with. Altogether, the preliminary and temporary exteriorization not only saves time, but will spare the patient much unnecessary buffeting while manipulations are conducted partly by feel in a confined space, the operator all the while being mindful of the ease with which the distended bowel may be perforated or otherwise damaged.

Any operative procedure is simple to carry out, while at the end no difficulty need be experienced in suturing the abdomen, with minimal risk of wound disruption.

The change in the intestine after effective decompression is most gratifying. Gone is the blue, congested, unhealthy appearance. Instead there are pink, collapsed, though admittedly somewhat œdematous, coils (*Fig.* 234).

The advantages of such decompression do not end with the operation, as there is considerably less post-operative morbidity and a smoother all-round progress. As was mentioned previously, it is easier to prevent the onset of distension than to treat the established case. If, after the patient is returned to the ward, an indwelling gastric (Ryle's) tube is regularly aspirated, there need be no fear of distension recurring.

Up to the present time the author has used the above method in 43 cases. The only feature which these cases had in common was that they were all long-standing low small-gut obstructions with gross distension, precisely the type of patient who is very ill and in whom a great deal of future morbidity is to be anticipated. Where only moderate distension has been encountered, simple relief of the obstruction without operative decompression has been practised. There has been only one death in this series, and this was a woman aged 77 years who was already moribund from general peritonitis following perforation of the ileum by a hair-ball.

All but 2 of the cases mentioned were suffering from mechanical small-bowel obstruction. In the remaining 2, paralytic ileus had been present for 12 and 14 days respectively following peritonitis. Neither had responded to conservative measures and

both were therefore decompressed by suction enterotomy, with successful results. On the basis of only 2 cases, it is too early to advocate this method in the treatment of resistant paralytic ileus, but it certainly has possibilities.

Of the mechanical obstructions, it was the rule for normal peristalsis to be resumed within 48 hours. All surgeons will recall many anxious post-operative hours in long-standing low small-bowel obstruction treated without operative decompression. Because of the very high survival-rate and the paucity of post-operative complications, the series quoted above can only be a significant recommendation for the routine use of operative decompression where gross distension is encountered.

SUMMARY

1. Distension continues to be one of the main factors contributing to the high mortality and morbidity rate in small intestinal obstruction.

2. The long intestinal tube is a valuable device, but is not a substitute for operation. Furthermore, the difficulty often experienced in successfully passing such a tube is exhausting to an ill patient, and its use in the advanced case is therefore not recommended.

3. Early operation is urged in non-strangulating mechanical low small-bowel obstruction after a preliminary restoration of fluid and electrolyte balance.

4. The desirability of surgical decompression of the distended intestine is discussed.

5. An instrument has been described and a technique evolved which are ideal for the purpose.

The author wishes to thank Mr. D. Lincoln Lewis, Mr. J. A. Baty, Mr. A. Glandon Williams, and Mr. J. A. Russell Johnson, of the Royal Salop Infirmary and Copthorne Hospital, Shrewsbury, for allowing him to operate on their cases. Also he wishes to express his gratitude to Mr. D. Lincoln Lewis for his encouragement, and to Professor F. A. R. Stammers and Mr. R. K. Debenham, of the Queen Elizabeth Hospital, Birmingham, for their advice and critical appraisal of the manuscript. Finally, thanks are due to Mr. Ross for the photographs and to Miss S. Turner for typing the manuscript.

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A CASE OF 'LIMY BILE' CAUSING OBSTRUCTIVE JAUNDICE

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THE presence in the gall-bladder of excessive amounts of calcium carbonate, either in a pure form or mixed with smaller amounts of calcium bilirubinate, is rare, but several cases have been reported from time to time. Volkmann presented a report in 1926 calling the condition 'Kalkmilchgalle' or milk of calcium bile. The term 'limy bile' appears to have been coined by Knutsson (1933).

Small amounts of calcium carbonate may be found on the surface of stones blocking the cystic duct, but it is much rarer to find a large amount in the gall-bladder and rarer still to meet a case in which obstructive jaundice is a feature.

CASE REPORT

Miss O. M., aged 18 years, was first seen as an out-patient on April 14, 1957, and gave a history of irregular recurrent attacks of epigastric pain dating back to the age of 7 years. The attacks were unrelated to meals and varied in intensity from a soreness to a severe ache lasting from two hours to two days. The pain did not radiate and there was no associated nausea, vomiting, anorexia, or bowel upset. No member of her family had biliary disease and there was no previous personal or family history of repeated fractures.

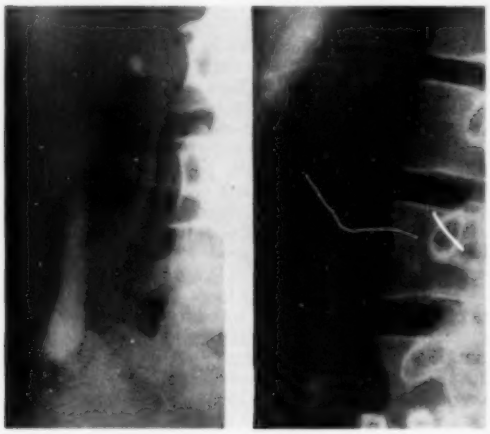
Apart from slight epigastric tenderness, no abnormal features were noted on investigation, and a plain radiograph of the abdomen was performed preparatory to a cholecystogram. *Fig.* 235 A shows this radiograph with its interesting shadow of an opaque gall-bladder and a further opacity higher up in the abdomen, possibly in the cystic duct.

An intravenous cholecystogram showed the outline of normal hepatic and common bile-ducts and a suggestion of some dye entering the gall-bladder. The separate opacity appeared to be in the region of the cystic duct (*Fig.* 235 B).

The day following the cholecystogram (ten days prior to admission to hospital) she developed a constant boring type of epigastric pain which radiated to the tip of the right scapula. It became worse about an hour after meals and also on movement. One day before admission the

pain became more intense and she vomited occasionally. Dark urine and pale stools were noted but the patient did not have attacks of shivering.

ON EXAMINATION.—She was pale and thin, and appeared to be in obvious distress. Her temperature was subnormal and the pulse-rate was 60 per minute. There was mild jaundice and marked upper abdominal tenderness, particularly in the right subcostal region; the liver edge could be felt for an inch or so below the costal margin.



A B

FIG. 235.—A, A plain radiograph shows the gall-bladder outlined by the contained calcium carbonate. An opacity is seen higher, possibly in the cystic duct. B, An intravenous cholecystogram shows some filling of the gall-bladder and an outline of the hepatic and common bile-ducts.

Pale fæces were present in the rectum and the urine was dark, containing bile-salts and pigments.

During the four days prior to operation the pain became increasingly severe, necessitating regular injections of pethidine. The pulse and temperature both remained within normal limits. Pre-operative intramuscular injections of vitamin K were given.

Further investigations showed increased calcification in the costal cartilages (*Fig.* 236) and the following blood chemistry was performed:—

Serum-bilirubin: Direct reaction positive. Total bilirubin, 3·5 mg. per 100 ml.

Serum-calcium: 11 mg. per 100 ml.

Serum inorganic phosphate: 4·5 mg. per 100 ml.

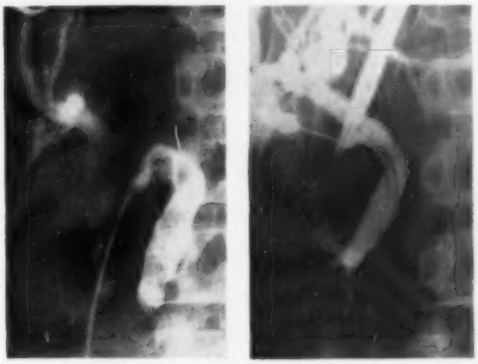
AT OPERATION.—Performed under general anæsthesia on April 30, through an upper right paramedian incision. A healthy, low-lying liver was seen on opening the peritoneum and a large gall-bladder, about twice the average size, was found on the visceral surface of the right lobe.

The gall-bladder had thick white walls and yielded 2-3 oz. of almost clear fluid on aspiration. In a prominent Hartmann's pouch was a mass of white calcareous material, almost solid, and measuring 1 in. × ¼ in. Contained in the centre of this mass were a few small, spiky pigment stones. The cystic duct was fully 1½ in. long and ¼ in. in diameter, was chronically inflamed and adherent to the side of the dilated common bile-duct, and its lumen was solid with the same calcareous material. The common duct was again chronically inflamed, ½ in. in diameter, and contained a mass of calcareous material from the level of the common hepatic duct to the termination of the common bile-duct.

A pre-exploratory cholangiogram was performed through the cystic duct with 35 per cent pyelosil and showed the outline of the cystic, common bile, and right hepatic ducts, but not much of the left hepatic duct. No dye was seen to enter the duodenum and filling defects could be seen (*Fig.* 237 A).

The common bile-duct was opened above the duodenum and was washed out thoroughly, explored with Desjardin's forceps, and palpated carefully. All obvious calcareous material was removed. Cholecystectomy was carried out, and before closure a post-exploratory

FIG. 236.—A further plain radiograph shows calcification in the costal cartilages as well as the outline of the gall-bladder.



A B

FIG. 237.—A, A pre-exploratory cholangiogram shows filling defects in the common bile-duct. B, A post-exploratory (T-tube) cholangiogram shows no residual defect and flow of dye into the duodenum.

cholangiogram performed through the T-tube showed passage of dye into the duodenum and no residual filling defects (*Fig.* 237 B).

POST-OPERATIVE PROGRESS was uninterrupted and the T-tube was removed on the fourteenth post-operative day, a cholangiogram being normal apart from the dilated biliary passages. At the time of discharge on the twentieth post-operative day there was no clinical evidence of residual obstructive jaundice.

Post-operative investigations were as follows:—

1. Culture of bile from common bile-duct and gall-bladder grew on aerobic culture non-hæmolytic streptococci.

2. Chemical analysis of 'limy bile' gave pure calcium carbonate.

3. Serum alkaline phosphatase, 8 King Armstrong units. Serum-calcium 12 mg. per 100 ml. Serum inorganic phosphate, 4·1 mg. per 100 ml.

4. Twenty-four specimen samples of urine for calcium (on a normal diet): 1st day: Total 24-hour urinary calcium output, 449 mg.; 2nd day: 422 mg.; 3rd day: 428 mg. (normal up to 300 mg.).

5. Histology of the gall-bladder showed features of chronic cholecystitis. (*Fig.* 238 shows the opened gall-bladder with adherent calcium carbonate.)

FIG. 238.—Calcium carbonate is seen adherent to the wall of the opened gall-bladder. (×⅔.)

6. Radiograph of hands and skull showed normal bone architecture.

Further calcium balance studies were contemplated but were not carried out as the patient emigrated to America. She has remained quite well in every way since the operation.

DISCUSSION

The most striking feature of this case is the presence of obstructive jaundice. None of the 7 original cases reported by Phemister, Rewbridge, and Rudishill (1931) gave a history of jaundice, and other observers, e.g., McCall and Tuggle (1942), say that it does not occur. Its rarity as a presenting symptom is confirmed by the fact that only 3 cases can be found in the literature. Cohen (1955) described a case in a 40-year-old female who had a three-months' history of biliary pain and two days' history of jaundice. The common-bile duct was dilated and contained creamy material, as did the hepatic duct. The gall-bladder was large and chronically inflamed and was removed at operation, the ducts being washed out and a stone found in the ampullary region. A further case was reported by Bergeret, Caroli, and Largarrenne (1947)—"Ictère par migration de bile calcaire dans le cholédochoque." This case was treated medically by cholagogues and recovered. The other case with jaundice was in an obese female of 20 years and was noted by Blanchet (1952). A pre-operative diagnosis of a cholecyst-duodenal fistula was made in this case, but disproved at operation. No analysis of the calcareous material was made and the common bile-duct was not explored, no mention being made of any abnormal features, despite the history of jaundice.

The rarity of migration of this calcareous material from the gall-bladder into the common bile-duct is possibly due to the presence of obstruction by a cystic duct stone in the majority of cases. The performance of the cholecystogram appeared to precipitate this migration just as in the case reported by Bergeret and others (1947). Presumably a certain amount of dye when allowed to enter the gall-bladder can stimulate it to intense contraction.

McCall and Tuggle (1942) reported a series of 15 cases and noted that the age and sex distribution of cases of 'limy bile' tend to be much the same as in the more usual cases of gall-stones. The present case appears to be the youngest patient to be operated upon and her symptoms date back some eleven years to the age of 7 years. Arianoff's patient (1953) was 24 years of age at the time of operation, but the attacks of pain dated back to the early age of 4 years.

The clinical picture of most cases appears to be mild and usually of a dyspeptic nature. Houghton (1952), in a case report and review of the literature, recalls that biliary colic is never severe when it occurs. In the present case the pain became increasingly severe, was prolonged, and necessitated regular doses of pethidine.

The occurrence of premature calcification of the costal cartilages has been noted in one other case of 'limy bile' by Arianoff (1953). His 24-year-old patient had a history of blue sclerotics and multiple fractures, and a strong family history of biliary disease. In none of the cases reported has a careful study of calcium metabolism been performed, and this would obviously be advisable in future cases, particularly if belonging to the younger age group. The present case suggests some anomaly in the calcium metabolism, and fuller studies may have confirmed this.

The exact mode of formation of 'limy bile' in the gall-bladder is not understood, although it is generally agreed that some form of obstruction to the cystic duct is necessary to its production. Phemister and his colleagues (1931) reported 7 cases of calcium carbonate gall-stones and calcification in the gall-bladder in which there was definite obstruction at the neck of the gall-bladder or in the cystic duct by a calculus. In Whipple's case the obstruction was due to a carcinoma of the gall-bladder. Wilkie's work (1928) on the bacteriology of cholecystitis is well known. He produced gravel and stones containing calcium and cholesterol by injecting streptococci intravenously or into the wall of rabbit gall-bladders in which the cystic duct had been tied. Phemister's experimental work on dogs also indicates that obstruction may play a part. He produced a coarse deposit of calcium carbonate by intramural injection of streptococci, ligation of the cystic duct, and the placing of a human gall-stone in the lumen of the gall-bladder. Cholecystography in this present case had suggested almost complete obstruction by a stone in the cystic duct region. Partial obstruction in addition may be caused by an unduly long cystic duct because of delayed or difficult emptying. The

cystic duct in this case was thickened and lengthy, as it was in the case reported by Majer (1952).

As a result of their experiments, Phemister, Day, and Hastings (1932) concluded that calcium carbonate was secreted by the gall-bladder wall in the presence of obstruction to the cystic duct. In the majority of cases of obstruction to the exit of bile from the gall-bladder a mucocele is formed and thus other factors must be involved. Whether or not partial or intermittent obstruction is more conducive to the formation of 'limy bile' is difficult to say. The intravenous cholecystogram performed in the present case showed a small amount of dye entering the gall-bladder, but in 6 out of Phemister's 7 cases oral cholecystography showed no concentration of the dye. In the seventh case only a plain radiograph was taken. Complete obstruction was obtained in his experimental work.

Johnston, Ravdin, Austin, and Morrison (1932) suggest that low-grade inflammation of the gall-bladder wall is also needed before calcium deposition can occur. McWhirter (1935) remarks that in the absence of inflammation cystic duct obstruction leads to a mucocele, whereas calcium is deposited in its presence.

Most writers argue that the calcium carbonate is precipitated out of the bile within the gall-bladder. Majer (1952) points out that normal bile contains twice as much calcium as does blood, namely 20 mg. per 100 ml. Maintenance of the proper proportion of cholesterol and bile-salts, he suggests, keeps lime in solution and alteration of these gives rise to precipitation. Chronic inflammatory change of the gall-bladder wall could easily do this, or by the same token a generalized calcium metabolism upset with a raised serum-calcium could do the same.

In other articles it has been suggested that the most important factor is the change in *p*H of the bile. The *p*H of normal liver bile is 8·2, i.e., on the alkaline side (Drury, McMaster, and Rous, 1924), and of the gall-bladder is 5–6. It has been supposed that obstruction to the cystic duct or disease of the gall-bladder may cause an alkaline swing of the *p*H of the gall-bladder, with resulting deposition of calcium carbonate normally present in the bile secreted by the liver.

The consistency and colour of the 'limy bile' will obviously vary according to the proportion of mucus and bile present. With a very weak solution true 'Kalkmilchgalle' will result. The more solid the calcareous material, the greater will be the likelihood of obtaining the interesting plain radiographic features. In the present case the picture obtained was a true outline of the gall-bladder similar to that produced in a normal cholecystogram.

This present case presents the possibility that the raised level of calcium excreted, together with partial obstruction to the cystic duct, gives rise to 'limy bile'. Radiography suggested a calculus in the cystic duct, and at operation an unduly long duct was found; either of these could alone have been the occluding agent. The premature costal cartilage calcification, elevated serum-calcium, and increased urinary calcium output (on a normal diet) indicated that a metabolic disturbance might be held partly responsible, but naturally further studies should be undertaken before a final opinion on this is expressed.

SUMMARY

'Limy bile' or calcium carbonate in the bile is a rare but recognized disorder affecting the gall-bladder. Less commonly does it present with obstructive jaundice, and a case is reported in which this occurred.

The young woman of 18 years in whom this disorder was found had premature calcification in the costal cartilages and possibly a disordered calcium metabolism. A plain radiograph of the abdomen gave a perfect outline of the gall-bladder and her attack of obstructive jaundice seemed to be related to the performance of a cholecystogram.

Many theories have been advanced in the past to account for calcium carbonate deposition in the gall-bladder and some of these are discussed.

Acknowledgements.—I am indebted to Dr. E. R. Cullinan and Mr. John P. Hosford for permission to publish this case, and in particular to Mr. Hosford for his advice and helpful criticism of this paper. Mr. Norman K. Harrison and his colleagues in the Photographic Department of St. Bartholomew's Hospital kindly made prints of the radiographs.

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OSTEOGENIC SARCOMA OF THE MANDIBLE

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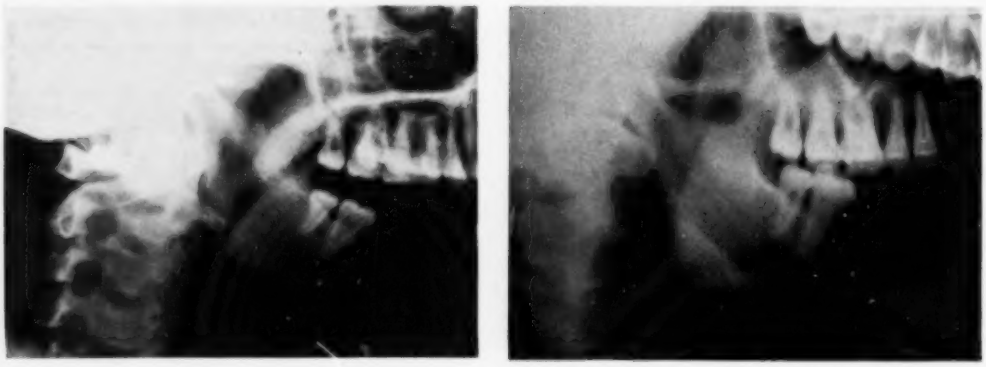
OSTEOGENIC sarcoma of the mandible is a well-recognized and not very uncommon tumour. Individual series ranging between 7 and 19 cases each have been recorded by Christensen (1925), Stout (1947), Coley (1949), Geschikter and Copeland (1949), Cade (1955), Sherman and Melamed (1955), Desaive (1955), and Dahlin (1957). There have also been many single case reports, and Richards and Coleman (1957) reviewed 13 in which photomicrographs had been published and added 1 further case of their own. The following case is of interest owing to repeated local bone recurrence despite radical resections of the mandible, followed by a satisfactory course for over two years after the whole of the lower jaw had been removed.

FIG. 239.—Radiograph of left mandible, showing area of destruction due to invasive tumour of bone.

FIG. 240.—Radiograph of right mandible, showing recurrence with 'sun-ray' spicules.



A B

FIG. 241.—Radiograph showing recurrence in mandibular remnant (B) compared with previously normal appearance (A).

CASE REPORT

HISTORY.—P. K., a married Indian woman, aged 25 years, was admitted to the Christian Medical College Hospital in January, 1955, with a history of a swelling in the left side of the face for four months and a sinus on the cheek which had developed after an operation at another hospital two months ago. The swelling recurred shortly after this and had gradually increased in size. She complained of severe pain and inability to open the mouth fully. Apart from amenorrhœa for the past five months, there was no abnormality in the rest of her history. She occasionally chewed *pan* (a mixture of betel leaf, areca nut, tobacco, and lime). There had been two pregnancies and both children were alive and well.

ON EXAMINATION.—The patient appeared in good general health. There was a swelling extending from the angle of the jaw to the side of the chin on the left side, measuring $3\frac{1}{2} \times 4$ in. A discharging sinus at the upper end of an oblique $1\frac{1}{2}$ in. long scar was seen over the centre of the swelling. On the inner aspect the swelling extended from the canine to the second molar tooth, expanding the gingiva. An irregular ulcerated mass projected into the mouth from the gingiva at the site of a recently extracted premolar. The swelling was warm, hard, and tender. A few small, mobile, firm glands were felt at the left submandibular area.

No other significant abnormality was found on general and special examination. B.P. was 120/80 and

hæmoglobin was 13·0 g. A radiograph of the left mandible showed the appearance of an invasive tumour of the bone (*Fig.* 239).

A biopsy on Jan. 20 showed scattered malignant epithelial cells with areas of extensive necrosis (E. W. G.).

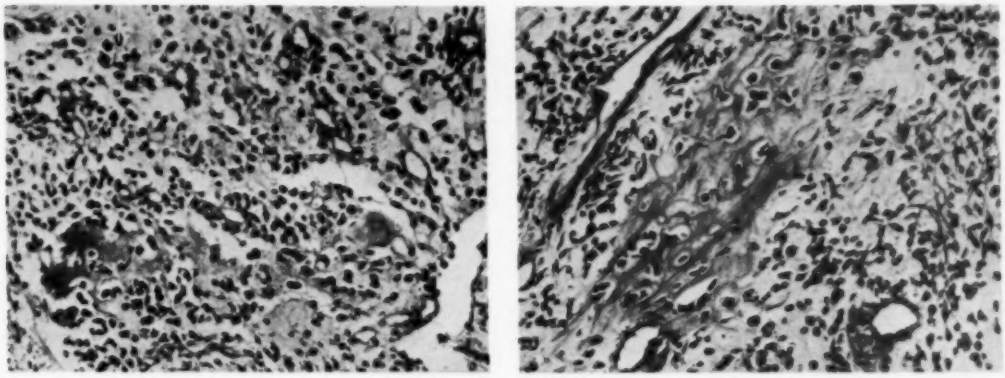


FIG. 242.—A, Low-power views. Shows typical appearances of osteoblastic osteogenic sarcoma including areas of tumour osteoid. H. and E. (× 123.) B, High-power view. Shows fully malignant pleomorphic stroma with tumour giant cells. H. and E. (× 256.)

A tentative diagnosis of atypical carcinoma of the gingiva with extensive bone involvement was made.

Left hemi-mandibulectomy with supra-omohyoid block dissection carried out on Feb. 2 (H. R. and D. M. H.). Recovery from this operation was uneventful and the patient was discharged on Feb. 16.

PATHOLOGY (E. W. G.).—The specimen consists of jaw with teeth, muscles, fat, skin, and lymph-nodes attached. The skin shows a scar of an old operation wound with a sinus at one end of it. There is also a large fleshy ulcerated growth arising from the alveolar margin. Histologically the appearances are those of osteogenic sarcoma, with sheets of pleomorphic malignant cells with dark nuclei and areas of osteoid and tumour bone.

PROGRESS.—The patient was seen again in July, 1955, with a two months' history of a rapidly growing painful swelling over the chin. On examination a shiny red, spherical 'tomato-like' swelling measuring 2 × 2 in. was seen projecting from the anterior end of the mandibulectomy scar. The swelling was hard, warm, and tender. It was fixed to the cut end of the right mandible and also to the overlying skin, but the mucosa was freely movable over its intra-oral portion. No neck glands were palpable. A radiograph of the right mandible (I. B. S.) showed "a dense tumour arising from its cut end. The tumour is rounded in shape and numerous fine bony spicules can be seen extending out from it" (*Fig.* 240). The appearances were typical of recurrent osteogenic sarcoma.

SECOND OPERATION.—This was carried out on July 23 (D. M. H.) and the recurrent growth, together with the attached skin and the horizontal ramus of the right mandible, was excised. The mandible was divided 3 cm. away from the posterior border of the growth. The operation was completed with a tracheotomy. Recovery was uneventful and the tracheotomy tube was removed on the sixth day after operation. Pathological examination (G. H.) showed similar appearances to those found previously with areas of tumour osteoid bone extending up to the epidermis in the area where the skin was fixed to the bone.

A post-operative course of deep X-ray therapy was started immediately and continued as an out-patient.

The patient was re-admitted on Oct. 11 complaining of a swelling in the right side of the mouth for the past month. She was having some difficulty on mastication but no pain. On examination there was marked keloid at the site of the previous operation scars. The cut end of the right mandible was thickened and the outer aspect of the alveolus was slightly expanded at this site. There was no pain or tenderness. Radiography of the right mandible (D. E. P.) showed an irregularity of the cut edge of the bone with some bony material extending forward into the soft tissues. The irregularity is best seen when compared with a radiograph taken in September (*Fig.* 241). Chest radiography showed no abnormality.

THIRD OPERATION.—It was decided that the excision of the remnant of the mandible should be combined with insertion of a bone graft in the left side of the cheek. The keloid scar on the left side was first widely excised and replaced with a rotation pedicle flap on Oct. 18. On Nov. 10 a 2½-in. section of rib was inserted on the left side and the remnant of the right mandible was excised together with a supra-omohyoid block dissection on this side (D. M. H.). The operation was completed with a tracheotomy. Recovery was uneventful and the tube was removed on the second post-operative day. The patient was discharged on Nov. 22.

PATHOLOGY (G. H.).—"Sections show dense fibrous tissue, small collections of osteoclasts, and other areas with sheets of pleomorphic and spindle-shaped nuclei. Occasional tumour giant cells are also seen. Spicules of calcification and osteoid are seen scattered through the

tumour. Salivary glands, lymph-glands, and skin show no evidence of tumour." (*Fig.* 242.)

Regular follow-up has been possible following this operation and her course has been satisfactory apart from development of a small stitch abscess in the left cheek in July, 1956. This was excised and biopsy showed inflammatory tissue only (G. H.). The patient was last seen on Jan. 5, 1958. (*Fig.* 243.) She was in good health and had no symptoms. Chest radiography on Jan. 5, 1958 showed no abnormality. Although unable to close the mouth completely owing to retraction of the chin, she could still manage to eat rice without undue difficulty. Further reconstructive surgery with insertion of a prosthesis for cosmetic purposes was advised, but the patient preferred to continue as she was without this.

A B

FIG. 243.—Appearance of patient on Jan. 5, 1958, approximately 2½ years after last operation.

DISCUSSION

The variable course of individual patients with osteogenic sarcoma, and the confusion that exists as to the exact histological diagnosis and classification of this disease (Coventry and Dahlin, 1957; Luck, 1957; Hatcher, 1957), makes prognosis at best a haphazard affair in this type of tumour. Widely differing five-year survival rates have been reported from different centres—e.g., 20 per cent (Badgley, 1949), and 5 per cent (Hatcher, 1957). Lichtenstein (1952) and Luck (1957) consider that chondrosarcomas have a better prognosis and that their inclusion in any series would increase the survival rate. However, Cade (1955) found no evidence that chondrosarcomas were less malignant than other osteogenic sarcomas. Dahlin (1957) has recently reported a 19 per cent five-year survival rate for osteogenic sarcoma, which he classifies into osteoblastic, fibroblastic, and chondroblastic varieties. In the osteoblastic group, in which, he says, no one could doubt the designation of osteogenic sarcoma, the survival rate was 15 per cent. Boyd (1955) has concluded that we have insufficient knowledge at present to give a reliable prognosis in this disease. It is not surprising, therefore, that there is a difference of opinion with regard to osteogenic sarcomas of the jaw.

Most authorities are agreed that the outlook is uniformly bad when osteogenic sarcoma arises in the mandible (Erich, 1952; Sharp, Bullock, and Hazlett, 1956; Ewing 1957). On the other hand there is good evidence that some osteogenic sarcomas behave more benignly when situated in the jaw than one would expect with similar tumours occurring in long bones (Simmons, 1942; Mallory, 1942; Hunter, 1954). Ivy (1949) and Desaive (1955) both included osteogenic sarcoma of the mandible amongst a series of slow-growing tumours of the jaw. The situation is best summed up by Thoma (1950), who states that there are two clinical categories when the mandible is involved. In one the tumours grow slowly and are osteoblastic. In the other the tumours are osteolytic, soft, and vascular, with a very poor prognosis. These criteria do not apply to tumours arising in other parts of the body (Calandruccio, 1956).

Widespread metastases are generally the cause of death in osteogenic sarcoma of the long bones. Local recurrences are uncommon, while the lungs are involved in nearly every patient at post-mortem examination (Cade, 1955). However, when the primary site is in the mandible local recurrence has been frequently reported (James, 1914; Slaughter, Roeser, and Smejkal, 1949; Thoma, 1950; Resch, 1955; Richards and Coleman, 1957), while lung metastases are infrequently mentioned. There were only 4 instances of pulmonary metastases amongst the 35 cases of sarcoma of the lower jaw (including 13 cases of chondrosarcoma) reported by Coley (1949) and the disease was still localized at death in 8 of the 15 fatal cases in his series. It is difficult to account for this in view of the widespread dissemination that

is found at autopsy when the primary is in one of the long bones, but the cause for this unusual behaviour may be related to the customary absence of red marrow in the mandible. This has been suggested as the reason for the rarity of metastases *to* the mandible in cases of breast carcinoma (Adair and Hermann, 1946). The frequency of local recurrence is probably related to disinclination on the part of the surgeon to remove much more than one hemimandible combined with difficulty in eradicating extensive local spread in the mouth and neck.

The satisfactory course after the initial rapidly repeated local recurrences in this case is of interest. The first operation that had been carried out before the patient came to this hospital was certainly inadequate, but the others were all radical and the bone was sectioned both times well beyond the gross extent of the tumour. Amputation through the affected bone is generally best avoided (Cade, 1952), but in the mandible radical resection really involves total mandibulectomy, and there is a natural disinclination to carry out this operation. The recurrences in this case were each time related to insufficient removal of apparently normal bone and not to inadequate excision of the extensions of the tumour into the tissues of the mouth and neck.

On reviewing the literature I found that the plan of treatment that was inadvertently carried out in this case was similar to that advocated by Ferguson (1940). He came to the conclusion that disarticulation or amputation should not be done early on in the disease, but that it should be preceded by excision of the lesion and deep X-ray treatment followed by radical removal during a 'quiescent' period. He states that should the tumour recur before the definitive operation, a second excision should be done and a waiting period again arranged after this until the final amputation or disarticulation.

It is difficult to understand the rationale of this line of treatment, although it resulted in a satisfactory outcome in this case. Further studies of tumour progression and host resistance are necessary before we can understand the meaning and significance of a 'quiescent phase' and its place in treatment. In osteogenic sarcoma of the mandible, however, total mandibulectomy may well be the treatment of choice, as the major cause of death is local recurrence. Should only half or three-quarters of the whole lower jaw be removed pathological evidence that the line of bone section has been through healthy bone and bone-marrow should be obtained. Whichever operation is carried out it should probably be preceded by a thorough course of supervoltage X-ray therapy, as advocated by Cade (1955).

The objections to total mandibulectomy are first removal of the tongue supports, and, secondly, interference with mastication. The first did not seem to inconvenience the patient at all, probably owing to the fact that the operation had been done in stages, between which the remaining tongue muscles had acquired new attachments. As for the second, it was quite amazing to see how the patient was able to cope with a large meal, mainly by breaking up the food between the tongue and the hard palate. Reconstruction of the soft tissues with insertion of a prosthesis (Erich, 1957) can easily be carried out as a final operation for cosmetic purposes.

The fact that this patient is still living and free from evidence of disease more than two years after her last operation is gratifying, but this does not mean that she is necessarily cured. Most deaths do occur during the first two years, but there are a few examples of late recurrence after a period of apparent cure. Dahlin (1957) states that most of the 4 per cent of cases with primaries in the long bones who died between five and ten years after treatment did so from the effects of their tumours.

SUMMARY

A case is reported in which it was necessary to carry out repeated excision of the mandible, culminating in total excision for rapidly recurring osteogenic sarcoma. The eventual outcome in this case was gratifying in that the patient has been repeatedly examined over a 2¼-year period since the last operation without evidence of re-appearance of the tumour either locally or elsewhere on the body.

A review of the literature has shown that the common cause of death in cases of osteogenic sarcoma of the mandible has been local recurrence, and widespread metastases have been infrequently reported. Total mandibulectomy associated with deep X-ray therapy may have a definite place in the treatment of osteogenic sarcoma of the lower jaw.

Addendum.—This tumour was not graded histologically according to the criteria of Price (1952, 1956), but with the limited material at hand it is probable that it belongs to either Grade II or III. It cannot be classified as a Grade I tumour. (Price, 1958).

Acknowledgements.—I would like to thank Mr. T. H. Somervell for his advice in the management of this case; and Dr. H. Ramamurthi, who carried out the first operation in this hospital with me. I would also like to thank Dr. Ida B. Scudder and Dr. D. E. Paterson for the X-ray reports; Dr. E. W. Gault and Dr. G. Hadley for the pathological reports; Dr. G. Hadley and Mrs. T. Macpherson for the production of the photomicrographs; and Mrs. M. Furness and Mrs. J. Parker for their help in the clerical work.

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GIANT ANTERIOR STOMAL ULCER

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INTRODUCTION

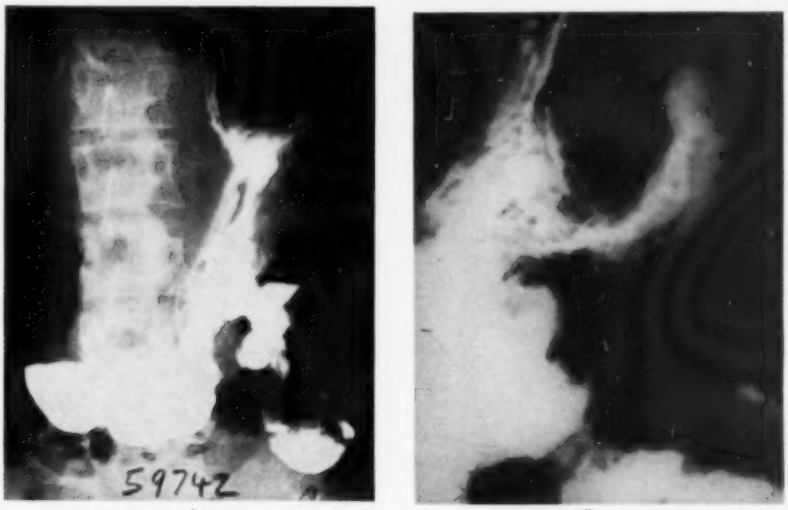
FIFTY years ago when anterior gastro-enterostomy was being frequently employed in the treatment of duodenal ulcer, chronic penetrating anterior stomal ulcer was a well-recognized entity. Paterson (1909) stressed the frequency of this condition and its association with the 'en-Y' type of anastomosis or with the combination of gastro-enterostomy and entero-anastomosis. Wright (1918) found that 36 of 74 anterior stomal ulcers reported in the literature had followed one of these two varieties of operation. He stressed the frequency of penetration of the abdominal wall and the formation of a palpable mass. Reports of this variety of stomal ulcer have become progressively less frequent since this time, owing to the infrequent use of these operative procedures in the treatment of duodenal ulcer. Lahey (1944) advocated the use of anterior gastro-enterostomy as a simple safe procedure in a fat patient with a short mesocolon and a distally situated duodenal ulcer, but emphasized that this should not be combined with jejunojejunostomy.

During the past year I have operated on 4 patients with large anterior stomal ulcers measuring between 4 and 15 cm. in greatest diameter. In all cases the previous operation had resulted in a stomal 'short circuit' (*Table I*). These cases are a reminder of the dangers of diverting bile away from the stoma of a gastro-enterostomy and an illustration of the size of the ulcers that can arise in such circumstances. This problem still has some relevance as, even in these days, entero-anastomosis between afferent and efferent loops is occasionally carried out for prolonged mechanical stomal obstruction after gastro-enterostomy or partial gastrectomy. Also Davey (1958) recently reported a large anterior gastrojejunal ulcer following a second gastro-enterostomy for a posterior stomal ulcer which resembled in many ways one of the cases described in this paper. Presumably, therefore, this inadequate procedure is still being carried out in Great Britain for fixed posterior anastomotic ulcer. The cases that I am reporting represent the direct result of contravention of fundamental surgical principles. All these patients were seriously incapacitated prior to surgical treatment of their stomal ulcers. In one (*Case* 3) fixed scoliosis to the affected side was directly attributable to the fibrosis of the abdominal wall and lower thoracic cage surrounding the base of a giant ulcer. (This measured 15 cm. × 12 cm. and is the largest stomal ulcer that I have been able to find in a review of the literature.) Another (*Case* 4) would have developed an external fistula if surgery had been delayed much longer. Fortunately surgical treatment presented no undue difficulty apart from the problem of wound closure in the last case. The fact that none of the previous operations was carried out in this hospital is of little credit to us, as a review of the

Table I.—CLINICAL DETAILS

CASE NUMBER, AGE, AND SEX	PREVIOUS OPERATIONS	SIZE OF ULCER	SITE OF PENETRATION
1. Male, 25	Anterior gastro-enterostomy with entero-anastomosis	4 cm. × 2 cm.	Muscles of abdominal wall
2. Male, 42	Anterior gastro-enterostomy with entero-anastomosis	5 cm. × 4 cm.	Muscles of abdominal wall
3. Male, 25	Anterior-gastro-enterostomy with entero-anastomosis	15 cm. × 12 cm.	Muscles of abdominal wall, diaphragm, and intercostal muscles
4. Male, 41	Anterior gastro-enterostomy for fixed stomal ulcer complicating posterior gastro-enterostomy	8 cm. × 6 cm.	Whole thickness of anterior abdominal wall, apart from skin and some subcutaneous tissue

records of this institution has not failed to show the occasional combination of jejunojejunostomy with partial gastrectomy or gastro-enterostomy during the past ten years. One of these patients died from



A B

FIG. 244.—Barium meal in *Case* 3 showing jejunal deformity and ulcer crater in erect view (A) and supine view with compression (B).

afferent-loop obstruction and another has been treated elsewhere for stomal ulceration.

CLINICAL DETAILS

Lack of periodicity of symptoms and absence of any clear relationship of pain to food were common features which are also characteristic of penetrating duodenal and gastric ulcers. Two cases complained of occasional vomiting, but this was not of the marked obstructive type which had preceded the first operation for duodenal ulcer in 3 of the 4 cases. The patients were all in poor general condition and 1 was severely anæmic (hæmoglobin, 6 g. per cent). In all, a mass was felt and this was fixed to skin, with œdema and induration of the skin over the mass in 2 cases. Barium-meal examination was a valuable adjunct to diagnosis (*Figs.* 244, 245). The sizes of the ulcers and the degree of penetration are indicated in *Table I*. The previous operations that resulted in these ulcers are also shown. None of these was carried out in this hospital. Photographs of the ulcer craters in the last two cases are shown (*Figs.* 246, 247). All the ulcers were deeply penetrating and presented interesting individual problems at operation.

Antecolic partial gastrectomy with removal of a portion of deformed jejunum at the site of the ulcer was carried out in each case. The ulcer base was left in situ on the inner surface of the abdominal or chest wall. The only difficulty encountered was in *Case* 4. At operation the incision was planned to skirt the left side of this ulcer, but the crater was inadvertently opened into at its left edge (*Fig.* 247). The peritoneal and muscular defect in the line of the abdominal incision had to be repaired by a free fascial graft in this case. Despite the inevitable soiling, primary wound healing was obtained. The post-operative course in all cases was uneventful and the longest post-operative hospitalization was fifteen days.

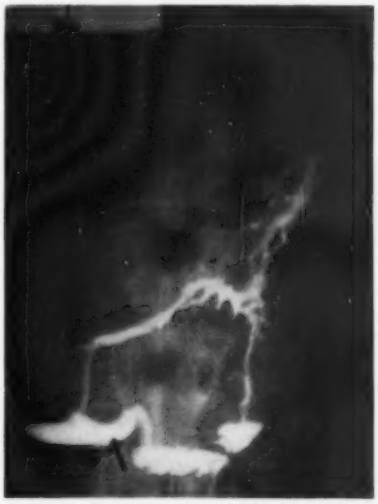


FIG. 245.—Barium meal in *Case* 4, showing stomal ulcer crater and jejunal deformity.

DISCUSSION

Aetiology.—The cause of gastrojejunal ulcer must be linked up with the cause of the original duodenal ulcer—namely, an increased parietal cell population of the stomach (Card, 1952; Cox, 1952). However, the formation of stomal ulcer will be enhanced if the operation designed for the treatment of duodenal ulcer results in: (1) Antral hyperactivity (Dragstedt, Woodward, Oberhelman, Storer, and

Smith, 1951; Thal, Perry, and Wangensteen 1957); or (2) Diversion of bile and pancreatic juice away from the site of the anastomosis (Wright, 1934).

Bile was short-circuited in these cases by jejuno-jejunostomy in 3 cases and by a second gastro-enterostomy in 1 case (*Table I* and *Fig.* 248). The addition of entero-enterostomy to gastro-enterostomy in many ways simulates the Mann-Williamson operation in the dog and results in a significantly higher anastomotic ulcer rate than gastro-enterostomy alone (Wright, 1934; Ivy, Grossman, and Bachrach, 1950). Some degree of antral hyperactivity was also probably present in all these cases, as the stoma was always found to be in the body of the stomach and not in the antrum. Oberhelman and Dragstedt (1955) have recently shown that a high-lying gastro-enterostomy results in a prolongation of gastric secretion which is not prevented by adequate vagotomy and can only be attributed to antral stimulation. The combination of vagotomy with an anterior gastro-enterostomy placed close to the pylorus, as advocated by Tanner (1958), is a useful operative procedure, as the neurogenic phase of gastric secretion is eliminated and the antrum remains unstimulated. This method is technically simpler than carrying out a juxtapyloric posterior retrocolic anastomosis. It should never be combined with a jejunojejunostomy.

FIG. 246.—View at operation in *Case* 3, showing portion of giant stomal ulcer crater.

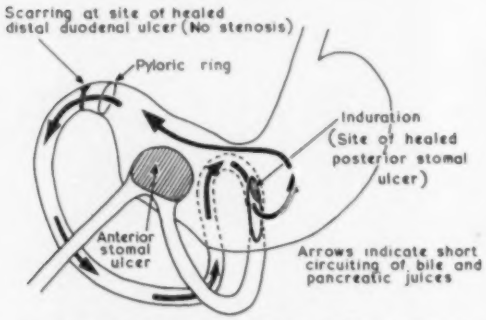

Scarring at site of healed distal duodenal ulcer (No stenosis)
Pyloric ring
Induration (Site of healed posterior stomal ulcer)
Anterior stomal ulcer
Arrows indicate short circuiting of bile and pancreatic juices

FIG. 248.—Diagram indicating the probable ulcerogenic effect of the operations that had been carried out in *Case* 4, resulting in the formation of a large stomal ulcer anteriorly.

Size of Ulcers.—Gastric ulcers are classified as true 'benign large' ulcers, or 'giant' ulcers, when the smallest diameter in the X-ray is 3 cm. or more (Jennings and Richardson, 1954), or when the largest diameter in the excised specimen measures 4 cm. or more (Turner, Dockerty, Priestley, and Comfort, 1957). If the same criteria were applied in these cases, all would come under the 'giant' category. However, apart from this factor of size, there is little similarity between these large stomal ulcers and giant gastric ulcers. The latter are usually associated with hypo-acidity or achlorhydria (Turner and others, 1957), and have a definite tendency to heal with medical treatment (Jennings and Richardson, 1954). High acid levels were found in 3 of these 4 cases and all showed remarkably little clinical response to medical treatment. The constant movement of the abdominal and chest wall undoubtedly contributed to the intractability and large size of these lesions. Pain was aggravated by walking in *Case* 3, and this association of pain with movement has been referred to by Wright (1918) and Walters (1948). Unusual reference of pain may also occur (Pickering, 1952).

FIG. 247.—Close-up view of operation in *Case* 4, showing major portion of ulcer crater occupying right half of photograph. The crater is seen to be continuous with the left margin of the incision above the Allis forceps as it was opened into in this region.

When there is wide involvement of the abdominal wall an extensive malignant growth may be simulated. The previous history and marked tenderness on examination help in the differentiation. Radiology should be more helpful in these cases than in the diagnosis of posterior stomal ulcers, where less than 40 per cent of craters are visualized (Walters, Chance, and Berkson, 1955). Craters were seen in each of these cases associated with marked jejunal deformity (*Figs.* 244, 245), and a correct pre-operative diagnosis was made.

SUMMARY

Details of personal experience with 4 cases of large penetrating anterior stomal ulcers are given, including 1 case in which the ulcer measured 15 cm. × 12 cm. This case is the largest stomal ulcer that I have been able to find records of in the literature. The size of the lesions and the intractability to medical treatment are probably both related to the constant movement of the ulcers with the abdominal and chest walls. Even in these days, it is necessary to stress the dangers of the combination of gastro-enterostomy with entero-enterostomy or another gastro-enterostomy in the treatment of duodenal ulcer, and these cases are an illustration of the size of the ulcers that can result in these circumstances.

Acknowledgements.—I would like to thank Dr. Ida B. Scudder and Dr. D. E. Paterson for the X-ray reports; and Mrs. T. Macpherson, Mr. S. Sigamoni, and Mr. V. Guiry for the drawings and photographs; also Mrs. M. Furness and Mrs. J. Parker for their help in the clerical work.

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TRAUMATIC DISLOCATION OF THE HIP IN CHILDREN*

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TRAUMATIC dislocation of the hip in children has always been considered a relatively rare lesion, though undoubtedly the paucity of references in the literature gives no indication of its true incidence. Blount (1955) remarks on its rarity, though Watson-Jones (1952) describes it as "no longer an unusual injury". It therefore appeared appropriate to review the literature on this subject and to record the experience at the Vancouver General Hospital with this injury in children up to the age of 16 years.

Choyce (1924) listed 53 cases from an extensive review of the literature and added 6 new cases; he apparently did not include 1 case reported by Campbell (1922). Subsequent years brought only isolated reports of single cases: Clarke (1929); Nicolaysen (1931), referred to by Banks (1941) and Goldenberg (1938); Chandler and Kreuscher (1932); Elmslie (1932); Glynn (1932), with 2 cases; Goetz (1934); Phemister (1934); Mauck and Anderson (1935); Haines (1937); Goldenberg (1938); Funsten, Kinser, and Frankel (1938), with 3 cases; and Kleinberg (1939). Banks (1941) considered the problem of aseptic necrosis of the femoral head following this injury and included in his review were 3 hitherto unrecorded dislocations in children, a fourth reviewed in some detail, and a fifth (his own case) presented. More recent papers have usually included children with adults in a large review: thus Thompson and Epstein (1951) mention 17 children in their survey of 204 cases; Paus (1951) includes 19 children and describes 1 case in a series of 75; and Stewart and Milford (1954) include 7 such cases out of 193 traumatic dislocations. Cases are described in the textbooks of Watson-Jones (1952), who describes 2 cases, and Blount (1955), who mentions 1 case; by Ghormley and Sullivan (1953) (and by Morrison in the discussion following their paper); and finally by Mason (1954), who reviews the literature and adds a single case. Thus mention or description of 129 cases can be found in the English literature, and the information gained from these cases is summarized in *Table I*.

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ANALYSIS OF REPORTED CASES

Age Incidence.—With an upper age limit of 16 years, the average age for 80 cases whose age is stated is 9·5 years; the commonest age by a slight margin is 6 years and the age range 4 to 11 years includes 58 of the 80 cases of known age. The youngest was 10 days, a traumatic dislocation apparently suffered during an obstetrical manœuvre, reported by Funsten and others (1938). There was only 1 other child under 1 year of age.

Sex Incidence.—There were 55 males and 23 females. Sex was not stated in 51 cases.

Table I.—DETAILS OF 129 CASES

AUTHOR	YEAR	NO. OF CASES	AGE	SEX	SIDE	MECHANISM OF INJURY	TYPE OF DISLOCATION	METHOD OF REDUCTION	RESULT
Choyce	1924	53 from literature 6 new cases							
Campbell	1922	1	11	M.	?	Struck by car while riding tricycle	Anterior (perineal)	Open reduction 12th day	Slight limp at 4 mth.
Clarke	1929	1	3	F.	?	Fell backward from brother's shoulders	Posterior	Two manipulative reductions required	Normal at 9 mth.
Chandler and Kreuscher	1932	1	14	F.	L.	?	Central	Closed reduction	Aseptic necrosis, with marked stiffness at 10 yr.
Elmslie	1932	1	7	F.	L.	Caught in elevator in hayfield	Posterior-superior	Closed reduction at 1 wk.	Aseptic necrosis pain and limp at 10 yr.
Glynn	1932	2	4	M.	?	Fell out of bed	Posterior (sciatic)	Bigelow's manœuvre at 5 d.	?
			7	F.	?	Fell off banisters	Inferior	?	Normal at 11 mth.
Goetz	1934	1	10	M.	L.	Knocked off scooter by car	Anterior (scrotal)	Bigelow's manœuvre	Normal though slight limp at 4 mth.
Phemister	1934	1	14	F.	?	?	Posterior, with fractured acetabular rim and floor	Closed reduction	Aseptic necrosis at 11 mth., good at 2½ yr.
Mauck and Anderson	1935	1	6	F.	R.	Fell 5 ft. to land on right knee	Posterior-inferior	Closed reduction	Normal at 3 yr.
Haines	1937	1	6	M.	L.	Fell off a moving sled	Anterior (?)	Simpson's manœuvre	Normal at 4 mth.
Goldenberg	1938	1	10½	M.	R.	Auto accident	Posterior	Closed reduction	Avascular necrosis at 15 mth. Normal at 2½ yr.
Funsten and others	1938	3	8	M.	R.	Hit by a car	Posterior	Closed red. at 3 hr.	Good at 2 yr.
			13	M.	R.	Riding in a car	Posterior	Closed red. at 3 wk.	Aseptic necrosis at 5 yr. Poor.
			10 d.	M.	R.	"Podalic version"	Posterior	Closed red. at 10 d.	Good at 5 yr.
Kleinberg	1939	1	16	M.	R.	?	?	?	?
Banks	1941	3 mentioned as hitherto undiscovered cases in literature							
		1 (Birsch-Hischfield, 1939)	12			?	Posterior (iliac)	Closed reduction	Aseptic necrosis at 3½ yr.
		1	12	M.	?	?	Anterior	Closed reduction	Aseptic necrosis at 15 mth. Good at 6½ yr.
Thompson and Epstein	1951	17	?	?	?	?	?	?	8 of 17 known to have *no* avascular necrosis
Paus	1951	18	?	?	?	?	?	?	?
		1	9	M.	R.		Posterior	Closed reduction at 10 d.	Normal function 16 yr. later, but X-ray evidence of aseptic necrosis
Watson-Jones	1952	2	?	?	?	?	Posterior	?	Aseptic necrosis at 4 mth.
			?	?	?	?	Posterior	?	?
Ghormley and Sullivan	1953	1	?	M.	?	?	Complicated by slipped upper femoral epiphysis	?	?
(in discussion)	1953	1	6	?	R.	Pushed off a log	Posterior	?	?
Stewart and Milford	1954	7	?	?	?	?	?	?	?
Mason	1954	1	1 yr. 11 mth.	M.	L.	Father tripped and fell on him	Posterior	Closed reduction, 8 wk. immobilization	Normal at 10 mth.
Blount	1955	1	21 mth.	F.	L.	Auto ran over her	Posterior	Closed reduction, 14 wk. immobilization	Well at 2 yr.

Side.—There were 30 right and 33 left hip dislocations. Side was not stated in 66; none was bilateral.

Type.—There were 61 posterior dislocations, 14 anterior, and in 51 the type was not stated. One dislocation was described as central, 1 as inferior, and 1 was considered to be a subluxation.

Mechanism of Injury.—The typical injury is said to be that of a weight falling on the back of a child in some sort of squatting posture (Glynn, 1932). Analysis of this information is quite inadequate, since in 109 instances the mechanism of injury was not stated. However, of the 20 remaining, only 2 were described as the typical injury; 9 were suffered in falls from a height, 8 in traffic accidents or similar accidents, and 1 was a "podalic version".

Interval before Reduction.—This interval varied from one hour to eleven years and in 68 instances was not recorded. Thirty-two dislocations were reduced within one week of injury, 29 were later than one week. There is a distinct tendency towards early accurate diagnosis and reduction in more recent cases.

Method of Reduction and Period of Immobilization.—In considering methods of reduction Choyce (1924) was able to conclude that manipulative reduction was always successful if carried out within the first 14 days, and in one instance was successful at 70 days. Of the 59 cases he presents, 31 closed reductions were successful and 16 open reductions were done; 4 underwent primary reconstructive procedures. Of the remaining 70 cases in the literature, few are reported in sufficient detail to allow any observations on method of reduction. Choyce (1924) makes no mention of after-treatment; of 14 cases in which after-treatment is recorded, immobilization ranged from 2 to 6 weeks, with restricted weight-bearing up to 14 weeks.

Complications.—

1. *Fracture.*—Dislocation in children is less likely to be accompanied by fracture, according to Blount (1955), and this would appear to be so in the 129 cases reviewed. Fractures of the acetabular rim and floor were noted in 2 cases (Phemister, 1934; Chandler and Kreuscher, 1932) and avulsions of the greater trochanter were recorded by Campbell (1922) and Goetz (1934). Fracture of the femoral neck was referred to by Funsten and others (1938) in 1 case and fracture of the femoral head by Thompson and Epstein (1951).

2. *Slipped Upper Femoral Epiphysis.*—Three cases of this displacement associated with traumatic dislocation of the hip were reported by Ghormley and Sullivan (1953), Watson-Jones (1952), and by Choyce (1924) (*Case* 10).

3. *Sciatic Nerve Injury.*—No such association in children was referred to in Choyce's review nor in subsequent cases in the literature.

4. *Recurrence.*—Apparently 5 of the 129 cases are known to have recurred. Choyce (1924) lists three: *Case* 18 recurred 6 weeks after operative reduction done four months after injury; *Case* 19; and *Case* 36, which redislocated after 10 days. Mauck and Anderson (1935) describe a single case recurring at 1 year, after a repeat injury. Goldenberg's (1938) case apparently became redislocated in plaster 1 week after reduction.

5. *Avascular Necrosis.*—Blount (1955) states that avascular necrosis of the head of the femur following traumatic dislocation of the hip is less common in children than it is in adults. Mason (1954), in his review, estimates its occurrence to be 9 of 89 cases, or 10 per cent, which compares favourably with the estimate of 10 per cent given by Nicoll (1952) in a review of 144 cases in adults. Stewart and Milford (1954), however, on the basis of 128 follow-ups, quote incidence figures of 15·5 per cent for closed reductions and 40 per cent for open reductions.

Of the 129 cases here reviewed, 12 are reported as developing aseptic necrosis or Perthes-like changes in the femoral head. Choyce's *Cases* 50 and 53 were detected at 1 year; that of Nicolaysen (*see* Goldenberg, 1938) at 1 year; Chandler and Kreuscher's (1932) at 6 months; Elmslie's (1932) at 7 months; Phemister's (1934), associated with acetabular fracture, at 11 months; Goldenberg's (1938), which recurred, at 15 months; Funsten and others (1938) at 5 years; Banks's (1941) at 15 months; Birsch-Hirschfeld's (*see* Banks, 1941) at 3½ years. Paus (1951) illustrates X-ray evidence of the change 16 years following the injury and Watson-Jones (1952) detected radiological changes at 4 months. Of Thompson and Epstein's 17 dislocations in children, 8 were known to have *no* aseptic necrosis.

In this small group of 12 cases with aseptic necrosis there appeared to be no outstanding common feature. Age ranged from 4 to 14 (average 10) years; there were 7 boys and 3 girls. Two had associated acetabular floor fractures (Chandler and Kreuscher, 1932; Phemister, 1934); 1 was a recurrent dislocation (Goldenberg, 1938); and 1 was anterior (Banks, 1941). Of 5 in which the interval between injury and reduction was stated, only 1 was reduced "within a few hours"; the others were reduced at 7 to 21 days.

6. *Traumatic Arthritis.*—This late complication has not been a feature of reports in the literature. Thompson and Epstein (1951) have recorded 1 such case.

NEW CASES

The records of the Vancouver General Hospital have been searched for cases of traumatic dislocation of the hip in children, aged 16 years or under, from 1936 to 1958. Seven such records were available, all admitted since 1949. It is suspected that the distribution of cases reflects the inadequacy of earlier records rather than a true yearly incidence. Information available on these cases is summarized in *Table II*.

The 7 cases ranged in age from 3·5 to 15 years, averaging 6·3 years. There were 5 boys and 2 girls; 3 involved the right hip, 4 the left. There was 1 anterior dislocation and the remaining 6 were posterior dislocations. Three patients suffered the classical mechanism of injury; each was stooping on all fours when another child fell on him. Two children suffered apparently severe injury in that they were involved in traffic accidents, but 2 suffered apparently minor trauma in a fall while running and a fall from a tricycle.

Reduction was accomplished within an average of 6·3 hours from the time of injury. General anæsthesia, with traction in 90° flexion of hip and knee, was a fairly standard form of manipulative reduction, though spinal anæsthesia was used in 1

case. In 2 instances intravenous skeletal muscle relaxant was added. No dislocation required open operative treatment.

Information on post-reduction management is incomplete, but does indicate a fairly consistent plan. One child (*Case* 3, G. L.), however, apparently had no period of immobilization or restriction of weight-bearing and her result was excellent. Of the remaining 6 cases, 4 were immobilized by plaster hip spicas, 2 by Buck's traction. This period of relative immobilization and non-weight-bearing ranged from 2½ to 7 weeks, followed by a period of partial weight-bearing lasting 1 week, so that full weight was carried at 5 to 8 weeks. In 1 instance (*Case* 4, J. H.) relative immobilization and non-weight-bearing were prolonged to 3 months by use of crutches and a leg sling, with full weight-bearing allowed at 5 months. His result, too, was excellent.

There were few immediate complications. In one (*Case* 4, J. H.), a small acetabular rim fragment was described; in one (*Case* 6, P. D.), a traffic accident and anterior dislocation, the contralateral sacro-iliac joint was also dislocated and an ankle injured. One 5-year-old boy (*Case* 7, W. C.) had no evidence of sciatic nerve injury on admission, but both motor and sensory signs developed in the hours prior to closed reduction. Recovery of the neurapraxia was complete within 3 weeks.

Five dislocations were available to follow-up examination over one year after injury (*see Table II*). All were classified as excellent results in that they had no complaints and had full function of the involved hip with normal physical findings. Three had radiographs taken at the time of follow-up examination and all of these were completely normal. There was no example of avascular necrosis and none of traumatic arthritis in this follow-up of 21 months to 9 years 2 months (average 4 years 8 months). The 2 remaining cases, too recent for one-year follow-up examination, were excellent at 6 and 4 months on the basis of clinical examination; no radiographs were taken.

Table II.—DETAILS OF AUTHOR'S CASES

CASE NO.	NAME	AGE	SEX	SIDE	MECHANISM OF INJURY	TYPE OF DISLOCATION	COMPLICATION	TIME BEFORE REDUCTION	METHODS OF REDUCTION	RESULT	PERIOD OF OBSERVATION
1	D. O.	3½	M.	R.	Stooping, jumped on	Posterior	No fracture or nerve injury	9 hr.	Traction in 90° flexion. General anæsthetic	*Excellent: full function. No complaints	1 yr. 9 mth.
2	R. M.	8	M.	L.	Stooping, jumped on	Posterior	No fracture or nerve injury	6 hr.	Traction in 90° flexion. Spinal anæsthetic with flaxidine	Excellent: full function. No complaints	4 yr. 5 mth.
3	G. L.	3½	F.	R.	Fell off tricycle	Posterior	No fracture or nerve injury	5½ hr.	General anæsthetic	Excellent: full function. No complaints	9 yr. 2 mth.
4	J. H.	15	M.	L.	Hit car while riding sled	Posterior	Small fragment off acetabular rim. No nerve injury	5 hr.	Traction in 90° flexion. General anæsthetic	Excellent: full function. No complaints	6 yr. 3 mth.
5	A. B.	3½	F.	L.	Fell while running	Posterior	No fracture or nerve injury	8 hr.	Traction and lateral pressure in flexion. General anæsthetic with anectine	*Excellent: full function. No complaints	1 yr. 10 mth.
6	P. D.	5½	M.	L.	Struck by a truck	Anterior	Dislocated right sacro-iliac joint with sprain-fracture right talus	6½ hr.	Manipulative reduction. General anæsthetic	*Excellent: full function. No. complaints	6 mth.
7	W. C.	5	M.	R.	Running, fell; another boy fell on him	Posterior	Sciatic nerve neurapraxia	5 hr.	Flexion to 90°, gently lifted in general anæsthetic	*Excellent: full function. No complaints	4 mth.

* No follow-up radiograph.

CONCLUSIONS

Certain broad conclusions are forthcoming from the 136 cases of traumatic dislocation of the hip herein reviewed. Children of any age, within the limits defined, are susceptible, though the injury becomes distinctly more common as the child begins to walk and becomes more venturesome. Boys predominate in a ratio of 2·5 to 1, but the dislocations are equally distributed between right and left hip. Posterior dislocations are of course commoner, in a ratio of 5 to 1. The mechanism of injury usually thought of as typical in children appears to be rather uncommon, in that only 5 of 27 suffered this type of stress. However, 3 of these were in the author's recent series of 7 cases.

The material available on interval before reduction, though limited, indicates a not unexpected trend to immediate diagnosis followed by manipulative

reduction within hours. Manipulative closed reduction under general anæsthesia has, as a result, become uniformly successful, there being no open reduction reported since Campbell's in 1922. Recommendations regarding period of immobilization and restricted weight-bearing, based on this series, are impossible since after-care received so little attention in the reports. Certainly there are fewer opinions expressed about this factor in the injury in children than there are in dislocations in adults. Of the author's small series of 5 cases, 4 are excellent with 2½ to 7 weeks immobilization and the fifth is excellent with no enforced immobilization whatsoever.

Fractures directly complicating the dislocation are indeed uncommon, there being a total of only 7 in the 136. Slipped upper femoral epiphysis occurred in 3 cases. In 3 of the fracture cases subsequent avascular necrosis occurred, so that the associated injury would appear to predispose to the later complication. Sciatic nerve lesions, too, are uncommon, *Case* 7 herein reported being the only occurrence in the entire 136 cases. It is interesting to speculate on this matter, since sharp bone fragments of associated fractures have been recently implicated in the production of the nerve lesion in adults.

Recurrence has not been seen since Goldenberg's 1938 case, and did not occur in the author's 7 cases. It is difficult to understand how this complication could occur in children without some dysplasia of the acetabulum when fracture is so uncommon.

The incidence of avascular necrosis, a change which one might expect to detect more easily in children than in adults, would appear to be 10 per cent. Whether or not the fact that no case in the author's series developed this change can be attributed to prompt and accurate reduction or to insufficient numbers is, of course, not certain. The figure corresponds exactly to that given by Nicoll for adults in 1952, though Stewart and Milford (1954) quoted higher figures of 15·5 per cent for closed and 40 per cent for open reductions.

No conclusions with respect to traumatic arthritis are possible on the basis of this review. It seems likely, however, that since fractures are uncommon in association with the dislocation and since avascular necrosis occurs in less than 10 per cent, traumatic arthritis can also said to be uncommon as a complication of traumatic dislocation of the hip in children.

SUMMARY

1. A total of 129 traumatic hip dislocations in children under 16 years of age have been summarized from the literature, and 7 new cases have been presented.

2. Early and late complications of this dislocation have proved to be uncommon. Avascular necrosis developed in less than 10 per cent.

3. The prognosis of this injury in children would appear to be excellent provided prompt and accurate reduction is carried out. No recommendations for after-care, based on material in this review, are possible.

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THE GASTRIC LESION OF DISSEMINATED EOSINOPHILIC COLLAGENOSIS OF BOUSSER

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Acute perforation of the stomach is usually attributable to peptic ulcer, injury, or growth. An exceptional and probably unique cause of gastric perforation is described in the following case-history.

CASE REPORT

A. F. C., a farm worker aged 47 years, was admitted to hospital with severe abdominal pain on Feb. 27, 1958; six weeks earlier his customary good health had been interrupted by daily epigastric pain induced by bending, eased by resting, and unconnected with eating or drinking.

Fig. 249.—Anterior perforated gastric ulcer from mucosal aspect.

On Examination.—He was in agony from severe abdominal pain radiating over the abdomen from the epigastrium. The belly wall was rigid and retracted and pressure at any point accentuated the pain. This seemed to be a clear case of perforated peptic ulcer. There was no past history of allergy, asthma, or other allergic complaint.

At Operation.—Laparotomy was made through an upper right paramedian incision under general anæsthesia. A large volume of turbid peritoneal fluid was sucked away until its source was found in a perforation of the anterior gastric wall close to the cardia, some 3 cm. from the lesser curvature. The perforation was the centre of a large area of rubbery thickening implicating the terminal œsophagus, cardia, and half the body of the stomach on both its aspects. The serous surface was smooth, grey, and opaque. Since the tissues around the perforation were brittle and rigid, making its closure impossible, a subtotal gastrectomy, including all the lesser curvature, was fashioned, not without difficulty because the tissues at the lower end of the gullet were only less friable than those around the perforation and the anastomosis here seemed precarious. Since the formation of a fistula was feared at this point of retrocolic jejunal union a soft rubber tube was placed down to the site in question.

Progress.—On the third post-operative day the drain discharged alimentary fluid and so a jejunostomy was immediately established under local anæsthesia and the drain was joined to an electric sucker. These devices were successful, and the patient's nutrition was sustained. The fistula closed by March 10, and he ate solid food from March 20. The jejunostomy was left for a few more days as a safeguard, but from that date the patient progressed without further setback.

Unfortunately, his blood-picture was not studied before March 6, when the histology of the gastric lesion was examined, and on that date the blood-picture was normal.

Specimen.—This represented practically the whole of the stomach (*Fig.* 249). The affected area was 3 cm. thick at its centre, and here the mucous membrane was interrupted by an ulcerous lesion like a volcanic crater, 4·3 by 2·5 cm. across its mouth. The apex perforated the serous coat through an aperture 1 cm. in diameter. Opposite this ulcer on the posterior wall lay a second ulcer eroding the mucosa to the muscle layer. Elsewhere the mucosa was hypertrophic or œdematous, the tissues were very vascular, but there was no sign of lymph glandular enlargement and no fibrosis in the adjacent omenta.

Microscopy.—"Nonspecific ulceration on a densely fibrotic, œdematous collagen base. Both this and the mucosa close to the lesion show pure eosinophil infiltrate. The smaller ulcer has the same structure. Vessels are normal. A section of mucosa from the pyloric region is virtually normal and devoid of eosinophils. No evidence of any worm infestation or similar disease found." (*Figs.* 250, 251.)

The gross appearance of this lesion was quite unlike any of the familiar gastric disorders and nowhere more so than at the ulcer site. Here the margins were neither terraced nor everted, and the walls tapered through the enormously thick gastric wall as a shaggy, cone-shaped depression bearing little resemblance to peptic ulcer or neoplasm.

DISCUSSION

Eosinophilic cells are present in most of the morbid processes which afflict the stomach, but in two conditions they are so abundant that their name has been applied to the disease. Vanek (1949) described 6 cases of a localized polypoid eosinophil granuloma, confined in each case to the pyloric region. Polayes and Krieger (1950) have reported a similar abnormality in the ileum. Kaijser (1937) gave an account of a diffuse form of eosinophilic gastric infiltration and two decades later Ferrier and

Davis (1957) state that less than 20 cases have been published. This distinction has been emphasized by Doniach and McKeown (1951) and also by Swarts and Young (1953) in their compendious report on this subject.

The clinical history does not permit certain diagnosis of this diffuse type, though the disorder might join the list of those other causes of low-grade, inconstant, irregular dyspepsia in the differential diagnosis. The condition assumes clinical urgency through its complications, and of these hæmorrhage seems the most frequent, manifested as hæmatemesis (Doniach and McKeown, 1951) or as melæna (Schneider and Dailey, 1948). The bleeding may derive from an acute erosion or from the oozing mucosa by gastrostaxis in the distal part of the stomach. Obstruction may be due to stenosis of the lumen or to rigidity of the stomach wall, and frequently causes vomiting. Finally, frank ulceration and perforation must now be added to the list of complications, though either development must be exceptional in a rare disease.

The disorder appears in the literature under a variety of titles—eosinophilic gastric granuloma, eosinophilic infiltration of the stomach, the gastric lesion of Loeffler's syndrome, allergic gastro-enteritis —according to the author's notion of the cause and

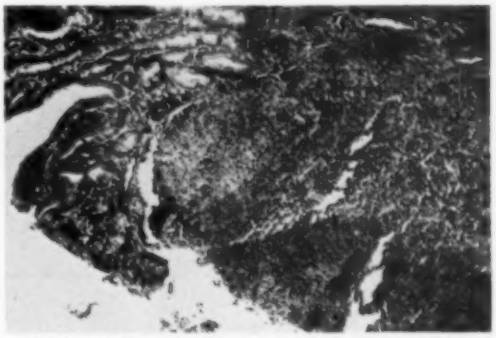

FIG. 250.—Muscularis and mucosa of body of stomach showing eosinophilic infiltration. H. and E. (× 25.)

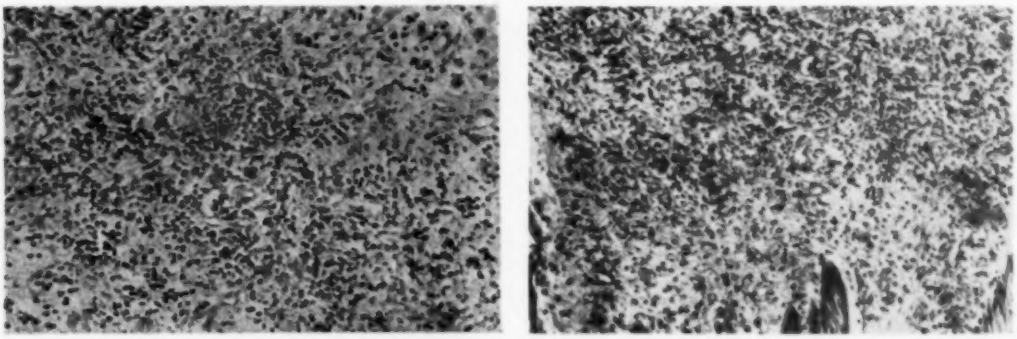

FIG. 251.—Eosinophilic infiltration of muscularis of stomach. H. and E. (× 50.)

pathology. The appearance of diffuse eosinophilic infiltration within the alimentary canal, though commonly found in the stomach, is by no means confined to it, for it has been found at most points between the pharynx and the colon and embryologically allied structures like the gall-bladder. Within the stomach the infiltration has been restricted to the antrum in all examples hitherto reported, but the pyloric valve provides no barrier to its spread into the duodenum and, as the present case shows, the eosinophil cells may proceed from the stomach into the gullet. The infiltration is endowed with the qualities of both Hydra and Proteus, for surgical removal of a local area does not insure against the reappearance of trouble elsewhere, and it may appear simultaneously at a number of sites widely separated by normal tissue. Reports on test-meal analyses show no distinctive changes in the secretion of hydrochloric acid.

Aetiology.—The deposition of eosinophil cells might well be accompanied by an increase in the circulating cells, and an eosinophilia has, in fact, been detected in the blood of some patients; indeed, it may occur in all patients, but an eosinophilia is apt to be ephemeral and therefore easily missed. Bousser (1957) provided the following causes of eosinophilia--Hodgkin's disease, allergic disorders, parasitic infection, cancer (especially with central necrosis), polyarteritis nodosa, chronic skin disease—but to this list he adds "disseminated eosinophilic collagenosis". Under this comprehensive title he includes certain ailments seen at all ages and in both sexes with eosinophilic manifestations—pulmonary infiltration, asthma, pericarditis and endocarditis, purpura, hypertension, hepatomegaly and splenomegaly, hemiplegia, cranial nerve palsies, and peripheral neuritis. Clinically the natural history may be brief, prolonged, or relapsing, and sometimes fatal, and the histological changes are typical of an allergic reaction. It seems reasonable to bring the alimentary eosinophilic infiltrations within this group of "disseminated eosinophilic collagenoses", and there is clinical evidence to justify it. Kaijser (1937), for instance, has reported 2 cases of eosinophilic infiltration of the small bowel in patients undergoing neoarsphenamine therapy for syphilis. Afendulis (1948), quoted by Doniach and McKeown (1951), described 5 cases of allergic gastritis in association with amœbiasis of the colon, while Ruzic, Dorsey, Hober, and Armstrong's (1952) patient had an eosinophilic count of 53 per

cent. Judd, Civin, and McIlhany's (1955) case of eosinophilic granuloma was asthmatic.

Treatment.—The morbid process can resolve completely (Swarts and Young, 1955), but perhaps the therapeutic feature which places the condition with the eosinophilic collagenoses is its response to steroid therapy. Ferrier and Davis (1957) were successful using ACTH, 40 units of a long-acting preparation for 14 days, and cortisone therapy has been effective. Partial gastrectomy has proved curative, but perhaps uncontrollable bleeding and perforation will prove to be the only compelling reasons for surgical treatment.

SUMMARY

1. A case of diffuse eosinophilic infiltration of the proximal end of the stomach with ulceration and perforation is reported.

2. This disorder is considered to be a gastric lesion of disseminated eosinophilic collagenosis of Bousser.

I am grateful to Dr. F. Kurrein for the pathological reports and for providing the photomicrographs.

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CHOLEDOCHUS CYST

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INTRODUCTION

CHOLEDOCHUS cyst, or congenital cystic dilatation of the common bile-duct, is a rare condition, first described by Vater in 1723, and occurring predominantly in young females. We wish to report 2 cases encountered within a period of a few weeks, and to add a note on a third case seen three years previously. All three patients came under the care of the same surgical unit, and make an interesting diagnostic series.

CASE REPORTS

Case 1.—P. W., a female child aged 12 years, attended the out-patient medical clinic in October, 1956, with a history of occasional attacks of abdominal colic of several years' duration, sometimes associated with vomiting. Old notes revealed that she had attended the surgical clinic in 1951, at the age of 6 years, with similar complaints; physical examination at that time had been normal and a provisional diagnosis of mesenteric adenitis was made. On the October, 1956, attendance, physical examination was again negative, as was also straight radiograph of the abdomen, and it was a surprise when she was referred back to the clinic about two months later, having virtually recovered from an attack of jaundice.

ON EXAMINATION.—Once again, apart from jaundice, clinical examination and straight radiograph of the abdomen were negative. Investigations showed Hb 99 per cent, W.B.C. 6600 per c. mm., E.S.R. 18 mm. in the first hour; urine negative for albumin, bile-salts and pigments, and urobilinogen-serum: bilirubin 2·9 mg. per cent.

The patient was admitted to the ward in April, 1957, on account of persistence of jaundice and nearly three finger-breadths of hepatic enlargement, but in addition there was now also a suggestion of an indefinite mass below the liver on the right side. A straight radiograph of the abdomen taken at this time was interpreted as normal apart from the enlarged hepatic shadow (*Fig.* 252). There was no complaint of pain and no abdominal tenderness on examination. Cholecystography in May, 1957

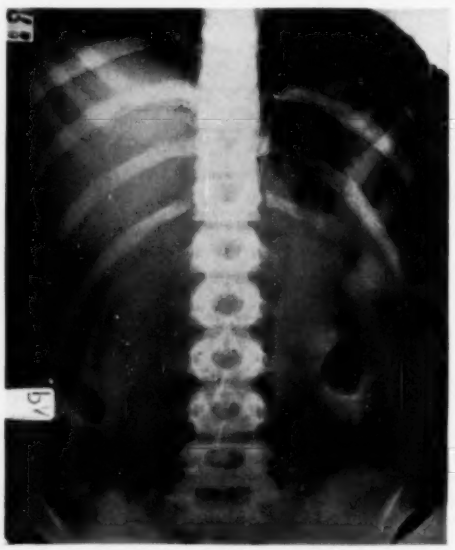

FIG. 252.—*Case* 1. Straight radiograph of abdomen, April, 1957. Although interpreted at that time as normal apart from the enlarged hepatic shadow, the absence of gas shadows in the right upper and mid abdomen is, in retrospect, almost certainly significant.

(*Fig.* 253) showed a non-functioning gall-bladder, and intravenous pyelography a few days later showed caliceal hydronephrosis on the right side (*Fig.* 254), without

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radiological evidence of enlargement of the right kidney. A barium meal in early June revealed an astonishing forward displacement of the whole duodenum (*Figs.* 255, 256). A diagnosis of choledochus cyst was made, and operation advised.

AT OPERATION.—Right upper paramedian incision. A large tense cystic swelling 4 in. (10 cm.) in diameter was found in the right upper abdomen with dilated right and left hepatic ducts running down into it. The duodenum and head of the pancreas were displaced forwards and downwards. The gall-bladder was moderately distended but otherwise healthy. The liver showed obvious naked-eye evidence of cirrhosis. The spleen was moderately enlarged, and the kidneys were apparently normal on both sides. The cyst was aspirated and 18 oz. (511 ml.) of clear bile removed, together with some biliary sludge. The upper jejunum was divided, the proximal end being anastomosed end-to-side to the distal loop, making a Roux-en-Y anastomosis, and the free end (6–8 in. long)

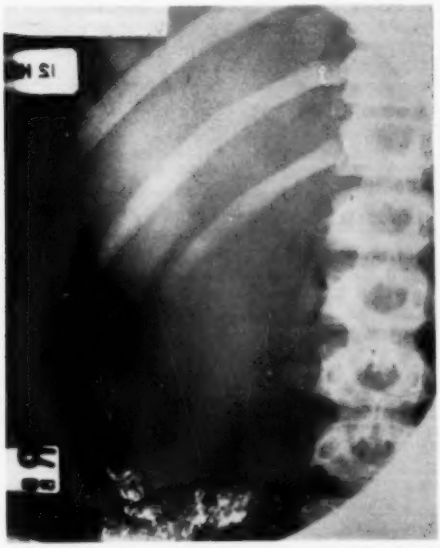

FIG. 253.—*Case* 1. Fifteen-hour telepaque film, May, 1957. The dye is in the right hepatic flexure of the colon which is at lumbar 4–5 level, and very probably displaced downwards by the choledochus cyst.

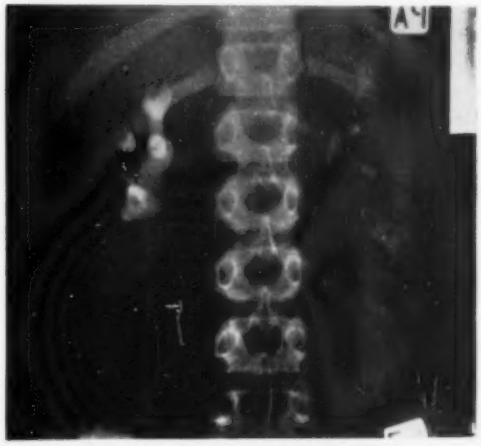

FIG. 254.—*Case* 1. Intravenous pyelogram, May, 1957. There is caliceal hydronephrosis on the right side.

FIG. 255.—*Case* 1. Barium meal, June, 1957. Right lateral view. Very marked anterior displacement of the duodenum, which is compressed against the anterior abdominal wall.

FIG. 256.—*Case* 1. Barium meal, June, 1957. Left oblique view. There is marked anterior displacement of the pylorus and duodenum against the abdominal wall.

was anastomosed to the front of the cyst. The abdomen was closed with drainage after liver biopsy.

POST-OPERATIVE RECOVERY.—Uneventful; discharged home at the end of June, 1957, with serum bilirubin 0·7 mg. per cent, and alkaline phosphatase 45 units per cent—the latter a lower figure than had been recorded for over a year. Biopsy of the liver showed evidence of marked biliary cirrhosis.

FOLLOW-UP.—In July, 1957, the patient was symptom-free, and gaining weight; serum bilirubin was 0·6 mg. per cent and alkaline phosphatase 28 units per cent. The liver was still palpable in the abdomen about three finger-breadths below the right costal margin. The patient has remained symptom-free up to the time of writing (August, 1958), but her long-term prognosis is thought to be poor because of the evidence pointing to liver damage.

Serial investigations on this case are shown in *Table I*.

Table I.—SERIAL INVESTIGATIONS ON *Case* 1

DATE	HB	W.B.C.	E.S.R.	S.B.*	URINE	A.P.†	RADIOGRAPHS	OTHER INVESTIGATIONS
Oct. 16, 1956	99	6600	18		Albumin: Negative Sugar: Negative		Oct. 16, 1956, chest and straight abdomen—N.A.D.	Differential blood-count normal
Oct. 17, 1956					C.S.U.: Normal			
Dec. 28, 1956	101	7800	27					Paul-Bunnell negative
Jan. 4, 1957				2·9			Jan. 4, 1957, Upper abdomen: "Shadow appears to be renal ? ptosed kidney"	Leptospiral agglutinations negative
March 1, 1957				5·1			March 1, 1957, Upper abdomen: "Moderate liver enlargement with downward displacement of kidney and hepatic flexure, and upward displacement of the right dome of diaphragm"	
March 29, 1957				5·5				Blood uric acid 2·8 mg./100 ml.
April 11, 1957	90	9200	59	6·0	Urobilinogen + + Bile ±	116·5		Serum proteins 8·8, albumin 4·9, globulin 3·9
April 17, 1957				4·0	Albumin: Negative Sugar: Negative	88·5		Bromsulphthalein test: within normal
April 23, 1958	86			5·6	Bile + +	109·5		
April 29, 1958			124	2·7		99		
May 6, 1958	88		95	3·8	Sp. gr. 1020 Albumin + : Bile + + Sugar: Negative	68		W.R. negative
May 13, 1958			100	4·1		53·5		
May 20, 1958			94	6·5		104·5		Serum colloidal gold 000000
May 27, 1958	90		81	3·6		100	May 27, 1958, Cholecystogram: "Non-functioning gall-bladder". I.V.P.: "Caliceal hydronephrosis, right"	Blood-cholesterol 175 mg. per cent
June 11, 1958	76			4·5	Bile + +	92		Blood group Rh. positive
June 12, 1958	Operation						June 11, 1958, Barium meal: "Whole duodenum displaced forwards against the abdominal wall, and of paper thickness"	Liver biopsy: "biliary cirrhosis"
June 17, 1958				1·5	Urobilinogen + + Bile ±	47·1		
June 25, 1958				0·7	Bile: Negative	45		
July 29, 1958				0·6		28		

* S.B. = serum bilirubin.
† A.P. = alkaline phosphatase.

Case 2.—F. J., a female aged 21 years, had been quite well apart from recent menorrhagia, and asthma since the age of 5 years, until four months before admission, when she had an attack of upper abdominal colicky pain followed by jaundice and vomiting. Her doctor thought she had virus hepatitis, and on a fat-restricted diet she was free from pain and most of the vomiting, but the jaundice persisted. The doctor found a subcostal swelling on the right side which he thought was liver.

ON EXAMINATION.—On admission to the West Norwich Hospital on Aug. 26, 1957, the patient was jaundiced, and a rounded swelling was felt in the right hypochondrium. The spleen was not felt and there was no ascites. The stools were pale and bulky and the urine contained bile-pigments. The results of investigations were as follows: Hb 80 per cent, W.B.C. 9200 per c.mm., E.S.R. 50 mm. in the first hour, serum bilirubin, 9·0 mg. per cent, alkaline phosphatase 59·8 units per cent, serum colloidal gold 000000; prothrombin time—normal, serum proteins 8·3 g./100 ml. total—albumin 4·7, globulin 3·6. A diagnosis of choledochus cyst was made.

AT OPERATION (Sept. 5).—Right paramedian incision. A large choledochus cyst was found with duodenum and

pancreas stretched over it, and adherent. The liver and gall-bladder looked normal, but the spleen was about twice normal size. The duodenum was stripped off and 34 oz. (816 ml.) of clear bile aspirated from the cyst, which was then anastomosed front-to-end to a retrocolic upper jejunal Roux-en-Y loop about 6 in. long. The wound was closed with a stab drain.

POST-OPERATIVE RECOVERY.—Uneventful; jaundice was relieved, and the patient was well enough to attend the plastic surgeon within the year for the treatment of another congenital abnormality—'undershot jaw' (micrognathia).

The unusual experience of encountering two cases within such a short period of time brought to mind a *third* which had occurred in the same surgical unit three years previously. This was the case of a 54-year-old female with a short history of epigastric pain and jaundice in whom cholecystography had shown a non-functioning gall-bladder, and who was admitted for cholecystectomy in May, 1955. At operation, a choledochus cyst was encountered, at least 2 in. (5 cm.) in diameter, but the nature of the condition was not recognized at the time. Both the cyst and the duodenum were opened and explored, but closed without further procedure, and no anastomosis was performed. The patient's post-operative recovery was stormy; she became jaundiced and pyrexial and developed an abscess at the site of operation, with subsequent biliary and fæcal fistulæ—complications which prolonged her stay in hospital to nearly three months before she was eventually discharged home with the wound healed.

DISCUSSION

Incidence.—Judd and Greene (1928) found only 1 case of choledochus cyst in 17,381 operations on the biliary tract. It follows that it must be unusual for 3 cases to occur in the same surgical unit in a period of 3 years in the absence of any factor of selection. All our cases were females; the condition is 4 times commoner in this sex; 75–80 per cent of reported cases are under the age of 25 years, and 60 per cent under 10. The condition seems to be commoner in Japan than in other countries.

Aetiology.—Fifteen theories of aetiology are discussed by Shallow, Eger, and Wagner (1943), but it seems to be generally agreed that there is a congenital weakness of the common bile-duct, possibly allied to Hirschsprung's disease or congenital idiopathic hydronephrosis.

Pathology.—The dilatation is usually confined to the supraduodenal part of the common bile-duct. The gall-bladder and common hepatic duct, or right and left hepatic ducts, which open into the top of the cyst, are usually not grossly dilated. They were fairly normal in size in these three cases. The common bile-duct is said to be constricted frequently, but in the third case, the only one of the three in which the inside of the cyst and the lower common bile-duct were explored, there was no obstruction to the passage of sounds.

Cysts have been described varying in size from that of a walnut to that of a full-term gravid uterus. The wall is usually thickened, as it was in our cases, and may be calcified. There may be evidence of inflammation, and carcinoma in the wall of a choledochus cyst has been described (Irwin and Morison, 1944).

Cholangitis and hepatic cirrhosis are common. There may be splenomegaly and ascites. Traumatic rupture of a choledochus cyst has been described (Blocker, Williams, and Williams, 1937; Dickson Wright, 1936).

Diagnosis.—These cases make an interesting diagnostic series in that the case of the 54-year-old woman was wrongly diagnosed before operation, and neither recognized at operation nor dealt with effectively, because the surgeon was unfamiliar with operative management of the condition; while the girl of 12 years was diagnosed before operation, but only after a long period of investigation; and the 21-year-old woman was diagnosed in a few minutes and on clinical grounds—the condition being uppermost in the medical attendant's mind.

To the standard triad of pain, tumour, and jaundice, one can frequently add fever. Hertzler and Maguire (1951) point out that in the older cases, careful history-making reveals previous episodes, usually with characteristic symptoms. It was our youngest patient who had the longest history. Gross (1953) found an average delay of three years between first complaint and diagnosis, and also stressed the point that the symptoms are usually intermittent.

Various radiological examinations may be helpful (Dickson Wright, 1935). The right kidney usually shows hydrocalycosis and hydro-ureter down to the level of the maximum diameter of the cyst (*see Fig.* 254). The cholecystogram may show filling in a comma-shaped gall-bladder, sitting on the top of the cyst, in the wall of which calcification may be visible. In our two cases in which cholecystography was ordered, the gall-bladder failed to concentrate the dye. However, telepaque outlined the hepatic flexure of the colon in *Case* 1 (*see Fig.* 253), showing it to be significantly depressed, and this was associated with absence of gas shadows in the right upper abdomen. The latter sign was evident in retrospect in the straight radiograph of April, 1957 (*see Fig.* 252), which had been passed as normal apart from hepatic enlargement.

The most dramatic radiological findings were in the barium-meal examination, which showed the duodenum displaced so far forward (*see Fig.* 256) that it was compressed against the anterior abdominal wall and flattened to such an extent that barium collected only at the angles (*see Fig.* 255).

Pre-operative diagnosis was made in 2 of our 3 cases. Shallow and others (1943), reviewing the literature, found a correct pre-operative diagnosis was made or suspected in only 12·6 per cent of cases, and that in many cases the diagnosis was not even made at operation, these patients usually dying.

It is obviously important to remember this condition and to consider it in persistent or recurrent jaundice in young females, especially if there is an abdominal swelling which cannot clearly be marked down as hepatic.

Treatment and Prognosis.—Dickson Wright (1935) was at one time of the opinion that operation was not indicated if symptoms from a choledochus cyst were mild. However, at that time the operative mortality was 80–90 per cent. As to the type of operation, he considered that choledocho-duodenostomy would always lead to cholangitis and eventual death, and

stated that results would only improve when the cyst was removed, the common bile-duct reconstructed, and the valvular mechanism of the bile-papilla thus retained.

It is generally considered to-day that choledochus cyst causes recurrent cholangitis and consequent liver damage, and should be treated by operation. Hertzler and Maguire (1951) recommend marsupialization of the cyst as a temporary measure in the acutely ill patient. They consider that anastomosis of the cyst wall to the duodenum (Gross, 1953; Ross and Carling, 1948) is liable to stasis and ascending cholangitis, and advocate excision of the cyst and direct anastomosis of the common hepatic or right and left hepatic ducts to the duodenum. Other methods of reconstruction after excision of the cyst include jejunal Roux-en-Y anastomosis (Keeley, 1948), cholecysto-duodenostomy (Davis, 1948), and anastomosis of the retained upper pole of the cyst to the duodenum (McLaughlin, 1946). Gross states that excision of the cyst is entirely unnecessary and suggests direct anastomosis between cyst and duodenum with or without a relieving cholecystostomy. However, it seems reasonable to attempt to reduce the risks of cholangitis (theoretically) by using a Roux-en-Y anastomosis as was done in two of our cases, and Maingot (1955) stresses that this operation, first performed by Priestley, should make a strong appeal to any surgeon encountering a choledochus cyst at exploration. Both our cases made an uneventful post-operative recovery, but the severe liver damage found in *Case* 1 makes eventual prognosis less hopeful.

SUMMARY

Two cases of choledochus cyst are described, with a note on a third. All occurred in the same surgical unit within a period of three years. Investigations, diagnosis, and treatment are discussed.

Acknowledgements.—We wish to thank Mr. Alan Birt and Dr. Batty Shaw for permission to publish these cases, and for encouragement in presenting them for publication.

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SOLITARY DISCOID KIDNEY WITH A SINGLE URETER OPENING INTO THE URETHRA

REPORT OF A CASE AND COMMENTS ON THE EMBRYOLOGY

BY D. GWYN MORRIS, CARDIFF

THE following is an account of a small child with a congenital solitary kidney and a single ureter having an ectopic opening into the urethra below the sphincter, together with an annotation of the operative procedures performed for its correction.

CASE REPORT

A little girl aged 7½ years was referred because of urinary incontinence since birth associated with retarded physical development and gross anæmia.

ON EXAMINATION.—She was a puny and anæmic child weighing 3½ st. with a height of 40 in. only. Her blood-pressure was 140/60 mm. mercury, and the retinal arteries were narrow and irregular. The spleen was palpably enlarged and she also had a genu valgum.

White blood cells were 5400 c.mm., blood-urea 150 mg. per cent, serum calcium 9 mg. per cent, plasma phosphate 5·8 mg. per cent, and the alkaline phosphatase 17 units per 100 ml. On two occasions bone-marrow biopsy yielded only small numbers of marrow cells. The urine contained albumin with a deposit of 2/3 leucocytes and it was sterile on culture.

Radiologically, no renal outline could be seen in either loin and an excretion pyelogram showed no concentration of the dye.

After transfusion of 350 ml. of concentrated red cells cystoscopy was performed. The appearances were disturbing as the cavity consisted of a saccular type of wall which could not be distended to ensure accurate visualization. The blood-urea now fell to 93 mg. per cent and there was an improvement in the patient's condition.

Cystography produced the appearances shown in *Fig.* 257.

AT OPERATION.—Under general anæsthesia a cystic mass was palpable in relation to the promontory of the sacrum and repeat cystoscopy showed no ureteric orifices in the urinary bladder. Laparotomy confirmed the presence of a solitary discoid pelvic kidney anterior to the sacrum. This was grossly hydronephrotic and lobulated. A very tortuous ureter with a diameter up to 3·5 cm. opened into the urethra (*Fig.* 258). There was no other kidney present and the uterus was normal.

The ureter was then dissected to its stenosed junction with the urethra and sectioned at this point. The lower

6 in. of the ureter were resected and the proximal ureter brought round the rim of the true pelvis extraperitoneally, along the line of the left common and external iliac vessels, and a mucosa-to-mucosa anastomosis was carried out to the anterior bladder wall in the prevesical space. An indwelling catheter was left in position for 7 days (*Fig.* 259).

FIG. 257.—Contrast dye injected along a catheter inserted via the external urinary meatus and passing immediately into the ureter.

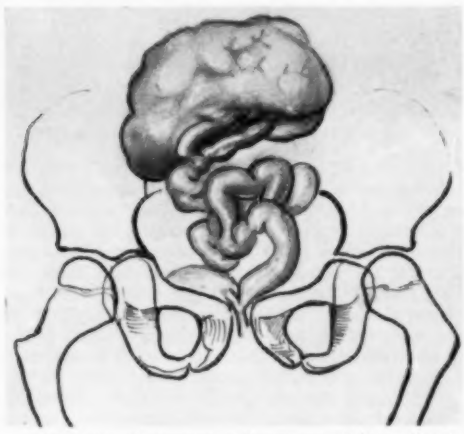

FIG. 258.—Appearances at laparotomy. Solitary discoid kidney with stenosed opening into urethra below the sphincter.

The immediate post-operative convalescence was stormy. The blood-urea rose to 230 mg. per cent, the patient had uraemic fits, and the serum electrolytes became grossly abnormal. Serum sodium was 134 mEq./litre, serum potassium 2·8 mEq./litre, serum chloride 109 mEq./litre, and carbon dioxide 11 mEq./litre. Urinary output then improved by the end of a fortnight and the blood-pressure and blood-urea levels returned almost to normal.

On discharge to her home a month after the operation the child could hold her urine for 1½ hours. There was no recurrence of anaemia and the blood-urea varied between 50 and 60 mg. per cent.

COMMENTARY

The chief interest, perhaps, is not the discoid kidney but the abnormal ureteric opening. Normally the urinary bladder is formed partly from entodermal cloaca and partly from the ends of the mesonephric ducts. After separation of the section from the cloaca the upper part of the cloaca joins the caudal ends of the mesonephric ducts and the associated ends of the ureteric diverticulæ arising from them, and these together give rise to the trigone of the bladder. In the female, the whole of the urethra is derived from the vesico-urethral or upper portion of the cloaca.

In this patient the bladder had no trigone; therefore there was no incorporation of the lower ends of the mesonephric ducts and the associated parts of the ureteric diverticulæ.

Figs. 258, 259 indicate that two ureters left the kidneys. If this is so, perhaps the midline kidney represents fusion of two kidneys with their separate ureters. The lower dilated portion could be the fused lower parts of the ureteric diverticulæ which have not become incorporated normally into the vesico-urethral portion of the cloaca to form the trigone.

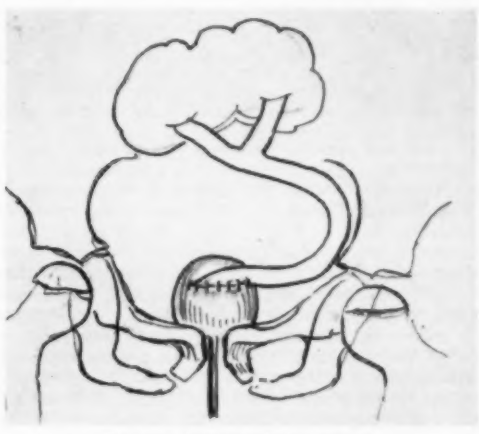

FIG. 259.—Post-operative appearances.

A STUDY OF SURGICAL MASKS

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According to Walter (1948), Mikulicz in 1896 was the first surgeon to use a mask to protect wounds from mouth bacteria. Some fourteen years later masks were regarded as optional accessories, although desirable for surgeons with colds (e.g., Waring, 1909; Rose and Carless, 1910). By the 1920's they had become an accepted part of the surgeon's uniform.

The realization in the late 1920's and early 1930's that the throat might be the source of hæmolytic streptococci responsible for puerperal infection led, not without protest (Solomons and Falkiner, 1937), to the use of masks by attendants during labour.

The regular wearing of masks as a part of the technique of wound dressing in this country probably followed the report of the Medical Research Council's War Wounds Committee and the Committee of the London Sector Pathologists (Medical Research Council, 1941), which again drew attention to the danger of infecting wounds with mouth organisms.

Fig. 260.—Showing volunteer's head inserted in table, and position of culture plates on floor of box.

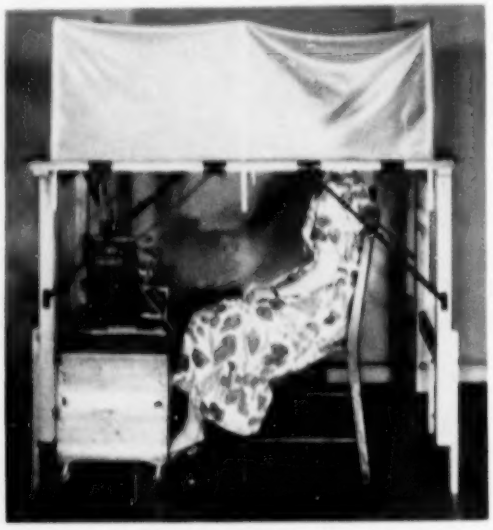

Fig. 261.—Showing canopy erected over volunteer.

Despite the experience of the last sixty years, there are still doubts as to the value of surgical masks in preventing infection. Hirshfeld and Laube (1941), in summarizing twenty-one papers, collected opinions which ranged from a firm belief in the efficacy of masks to an equally definite disbelief. Many authors think that during quiet breathing few, if any, bacteria are expelled from the mouth or nose. It is a matter of everyday observation, however, that during sneezing, and to a lesser extent during coughing and talking, particles are blown from the mouth and nose. Many of these particles can be shown to contain bacteria.

Surgical masks are presumably valuable if they shield the patient's wound from pathogenic bacteria in the surgeon's mouth and nose. This they may do by filtering bacteria from the air passing through them, or, being impervious, by deflecting the expired air so that it travels behind the head. The work which we describe was done to test surgical masks, and particularly to see how often staphylococci were shed by masked and unmasked nasal carriers.

METHODS

These studies were made possible by the co-operation of 129 volunteers, some of whom helped us more than once. Each volunteer sat for 15 minutes with his or her head from the neck up enclosed in a box 50 in. long and 18 in. high, 20 in. wide at the head end and 30 in. wide at the other. This was done by inserting the volunteer's head, without it touching the side, through a hole in a table (*Fig.* 260), sealing the gap round the neck by allowing a rubber diaphragm to spring back into place, and erecting over the volunteer a plastic canopy held up by the poles seen in *Fig.* 260 and fastened to the edge of the table by battens (*Fig.* 261). For each test approximately twenty 4-in. Petri dishes containing blood-agar were placed on the floor of the box (i.e., on the top of the table) in the positions shown in *Fig.* 260. Filtered air entered from a pipe behind the head and was sucked out at a rate of 1 cu. ft. per min. through an orifice 6 in. above table level and 2 ft. 6 in. in front of the volunteer. In some of the early runs this pipe was ½ in. in bore; in subsequent runs a pipe of 1-in. bore was used. This air was collected by a slit sampler. At the end of each run the humidity had usually risen by 25–30 per cent.

Half an hour before each test the interior of the canopy and the top of the table were wiped over with

a mixture of cetrimide (0·5 per cent) and chlorhexidine (0·05 per cent). The sides of the canopy were then shut, and air was drawn through the box at 1 cu. ft. per min. for 15 min.

Masks.—When masks were used the volunteer washed and dried the hands and put on a sterile cap and mask. Three types of mask were tested.

Filtration-type Mask.—A bucket-shaped mask into which the nose and chin fitted fairly snugly. It was made of four layers of gauze, each layer having 46 threads to the inch.

Tail-type Mask.—A deflexion mask made of closely woven cambric measuring 7½ in. × 8½ in. It was of double thickness so that a piece of paper could be inserted between the two layers. Attached to the mask was a tail of the same size and material. When worn, the nose, mouth, and chin were covered, and the tail hung down so that when a gown was worn it lay beneath the gown. The fit over the cheeks was loose.

Paper Mask.—A deflexion mask measuring 6½ in. × 5¾ in. which was made to be worn once and then thrown away. It consisted of an outer and inner layer surrounding a pad of cellulose wadding. It covered the nose, mouth, and chin, but the fit round the cheeks was loose. As supplied to us it could not be sterilized by heat without damaging it.

Tests.—Most of the tests were done with volunteers who were silent or who spoke quietly, as these two situations are those most often met at operation or during dressings. During silent runs the volunteer spoke only when answering an initial question as to comfort and when giving a humidity reading. In runs in which the volunteer talked an attempt was made to sustain quiet but continuous conversation. At the end of each run swabs were taken from the nose, from the underside of the tongue or the throat, and from the skin of the face below the mouth.

Table I.—Bacteria isolated from Plates on the Floor of the Box

Test		Medians of Average Colony Count per Plate			
Type	No.	Area			
		I	*II*	*III*	*IV*
Dummy	23	0·6 (0)*	0·8 (0)	0·8 (0)	0·5 (0)
No mask, silent	21	1·6 (0)	1·4 (2·3)	1·6 (0)	—
No mask, talking	27	5·0 (21·7)	1·3 (1·2)	1·7 (0)	2·0 (0)
Filtration mask, silent	19	1·8 (0)	1·7 (0)	2·5 (1·5)	1·5 (0)
Tail mask, silent	16	1·1 (0)	1·2 (0)	1·5 (0)	1·2 (0)
Paper mask, silent	9	1·0 (0)	2·0 (0)	1·2 (0)	1·0 (0)
Filtration mask, talking	25	1·8 (0)	1·3 (0)	2·5 (0)	1·0 (0)
Tail mask, talking	21	1·8 (0)	1·1 (0)	2·5 (0)	1·5 (0)
Paper mask, talking	10	1·7 (0)	1·7 (0)	2·4 (0)	1·2 (7)†

* Figures in brackets denote percentage of plates with 10 or more colonies.
† Based on only 1 plate out of 13 with 10 or more colonies.

Staphylococcal colonies were tested for coagulase production in a tube with plasma; all positive strains were submitted for phage typing. No attempt was made to identify other bacteria isolated.

RESULTS

We found that we were unable to go through the stages of lifting the canopy, introducing the volunteer's head, inserting twenty culture plates, lowering and clamping the canopy and then raising it again and removing the plates at the end of fifteen minutes, without introducing some extraneous bacteria. To follow this contamination twenty-three dummy runs were interspersed among the genuine tests. Each mimicked an ordinary run except that no volunteer was present.

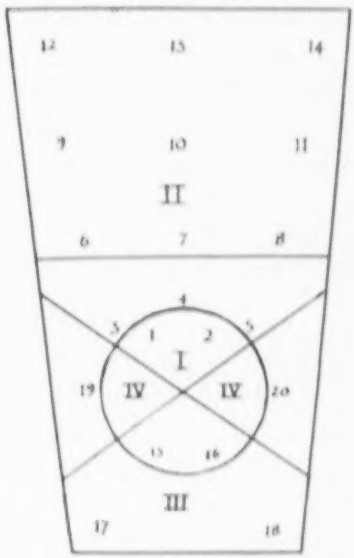

Fig. 262.—Plan of floor of box. Arabic numerals denote position of culture plates. Roman numerals show division of floor of box into areas used in *Table I*.

The first comparison was between dummy runs and tests in which unmasked volunteers spoke or kept silent. The results are given in *Table I*, in which the floor of the box has been divided into four areas, as shown in *Fig.* 262. The colony counts for the plates in each area have been averaged, the figures shown in the table being the medians of such averages for the different volunteers. In addition, in brackets after the medians the numbers of plates per 100 with 10 or more colonies have been given.

During the dummy runs there was a low and evenly distributed contamination of plates in all areas of the floor of the box. In the silent tests without a mask the number of organisms was approximately doubled in the first three areas, and in the second area, the front half of the box, 3 out of 133 plates (2·3 per cent) contained more than 10 colonies. The actual counts were 24, 31, and 27 colonies per plate and all three plates were part of a single test.

Talking increased the counts on the floor of the box and, as might be expected, the increase was chiefly in front of the mouth in Area I. Counts rose in two ways. As shown by the medians there was a general rise in organisms isolated, but in addition 21·7 per cent of the plates contained 10 or more colonies. In some the contamination was heavy, 3 plates containing an uncountable number of colonies. One plate had 195 colonies and 4 had between 40 and 50.

With one possible exception, there was little difference in the number of organisms isolated when silent volunteers wearing any of the masks were compared with silent unmasked volunteers.

Table II.—Bacteria isolated from the Air *

Test		Air Count (Colonies/15 cu. ft.)	
Type	No.	Range	Median
Dummy	11	8–22	16
No mask, silent	6	6–21	16
No mask, talking	11	12–46	24
Filtration mask, silent	19	11–44	22
Tail mask, silent	14	5–39	19
Paper mask, silent	9	8–41	31
Filtration mask, talking	22	7–43	23
Tail mask, talking	19	8–42	25
Paper mask, talking	9	11–36	20

* Based on counts with 1-in. bore tube only.

The effect of the mask, however, was clearly shown in tests in which the masked volunteers talked. All three masks reduced the number of organisms in front of the mouth to a level close to that found for silent unmasked volunteers, and perhaps more importantly, none of the culture plates in front of the mouth had more than 10 colonies per plate as compared with a rate of 21·7 per cent for the unmasked talkers. Not all the extra organisms put out during talking, however, were retained by the mask, as there was a rise in the counts behind the head (Area III) and presumably this can be accounted for by organisms which were deflected by the mask and carried backwards by the expired air.

The foregoing tests gave some indication of the performance of the mask during its first fifteen minutes of use. An additional thirteen tests with deflexion type masks were carried out to see if the efficiency of the mask dropped after longer use. Three of these tests were done after the mask had been worn in the laboratory for three hours. The remaining ten tests were done with members of a surgical team, who had worn one mask for an operating session of approximately four hours.

The combined results of these experiments suggested that after prolonged use more organisms were liberated, the medians of the average count per plate for the four areas of the floor of the box being 2·6, 2·3, 2·7, and 1·5 respectively. None of the plates, however, contained 10 or more colonies, and the results may not reflect failure of the mask, as in both sets of experiments the mask was lowered round the neck at intervals. As will be seen below, this practice carries with it the risk of contaminating the outer as well as the inner surface of the mask.

The results of the air counts (*Table II*) with or without masks showed a surprising degree of uniformity, and perhaps lack of effect of the mask in retaining smaller particles.

Shedding of Staphylococci by Nasal Carriers.—Staphylococci so clearly head the list of bacteria responsible for hospital infection that particular attention was paid to the liberation of staphylococci by carriers. The main results are shown in *Table III*. From 116 tests conducted with nasal carriers, 10 colonies of staphylococci of the same phage type as those carried by the volunteer were isolated; 5 of these came from plates on the floor of the box and 5 from the air. Four of the 5 colonies on plates came from unmasked subjects, as did 2 of the 5 air colonies. Thus, for the floor of the box, 4 colonies came from 39 runs with unmasked and only 1 from 77 runs with masked volunteers. The difference between masked and unmasked volunteers was not so marked in the recovery of staphylococci from the air, which was perhaps in keeping with the previous record of air counts (*Table II*), indicating that masks were less successful in retaining smaller particles. There was no suggestion that masks increased the rate of liberation of staphylococci by friction over the skin of the face.

Table III.—Isolation of Staphylococci of the same Phage Type as those Present in the Volunteer's Nose

Test		Colonies of *Staph. aureus* isolated	
Type	No. of Carriers	On Plates on Floor of Box	From Air
No mask, silent	18	1	1
No mask, talking	21	3	1
Mask, silent*	26	1	1
Mask, talking*	36	0	2
Miscellaneous†	15	0	0

* Combined result for three types of mask used.
† All tests with masks, some after use, some with coughing and talking.

By accident, evidence was obtained of another method of staphylococcal spread. One of the assistants was a skin and nasal carrier of a staphylococcus of a relatively uncommon and easily recognizable type. Approximately one culture plate in 60 of those on the floor of the box contained a colony of this organism. These staphylococci probably came from the assistant's arms and were shed during manipulations attendant upon inserting the volunteer and introducing the culture plates. It was of some interest that from 10 runs when this assistant was the volunteer, only one staphylococcal colony was isolated.

Finally, tests were done to see how often masks might be contaminated with staphylococci from the carrier's nose or face. In making these observations we had in mind the not unusual practice of wearing one mask throughout an operating session, or during half a day's work in a ward.

A total of 53 examinations was made of masks from 11 nasal carriers, all of whom were working in operating theatres. The masks were of the deflexion type, had been worn on an average for 4 hours, and between operations might have been lowered round the neck. Of the 53 masks, *Staph. aureus* was isolated from the inner surface on 33 occasions and from the outer on 21 occasions. It was not possible to be certain where the staphylococci on the outer surface came from, as frequently the paper in the mask which was responsible for deflexion had slipped out of place.

Throughout this work no Group-A hæmolytic streptococci were isolated from any of the culture plates on the floor of the box or in the air sampler.

DISCUSSION

As others have observed, the best way to prevent mouth organisms getting into operating-theatre air is not to talk, but although to keep silent may be ideal it is not always possible, neither is it always possible to change dressings in the ward without speaking. Our results suggest that the three masks we tested would fulfil their main purpose in protecting the patient's wound from many of the bacteria in the surgeon's mouth when the latter talked. Complete protection is not at present easy. Very approximately 40 cubic feet of air have to find a way in and out, through or around a mask, every hour. Increasing the thickness of the mask or improving the fit may lead to a point at which it becomes difficult to breathe. With deflexion type masks it seems probable that deflected expired air will inevitably carry some of the shed bacteria behind the head.

From the few colonies isolated the three masks we tested also appeared to reduce the number of staphylococci shed in what were probably heavier particles, and isolated on the floor of the box. The expulsion rate for staphylococci is clearly low, and our findings agree in this respect with earlier reports (Duguid and Wallace, 1948; Hare and Thomas, 1956). As the number of staphylococci needed to infect a wound is unknown, it is difficult to know the importance to attach to the occasional colonies we isolated from our carriers. By comparison with the much larger numbers of staphylococci which could be shed in other ways it seems unlikely that much additional attention to the structure and fit of masks would be rewarding.

In the operating theatre, therefore, there is a good case for retaining surgical masks on the grounds that they protect the area in front of the surgeon's mouth and probably restrict the number of staphylococci which are set free. The patterns we tried did not appear to increase the number by friction on the skin.

There is probably a case for retaining masks in wards, as long as it is realized that a used mask may be contaminated with the wearer's staphylococci. This does not matter when a fresh mask is worn for each dressing, but may become important if a single mask has to be used for a number of dressings. This practice is not unusual, the mask being hung round the neck, or even put in the pocket between use. Handling a worn mask entails the risk of contaminating the hands, and if a fresh mask cannot be provided for each dressing, care must be taken to wash the hands every time the soiled mask is touched.

SUMMARY

Tests have been carried out with three patterns of surgical mask. All were found to protect the area in front of the mouth from many of the wearer's mouth and nose organisms, and there was some evidence that they reduced the number of staphylococci shed.

It is suggested that there is a good case for the retention of surgical masks in operating theatres, and in the wards if a fresh mask can be provided for each dressing. If a fresh mask cannot be provided the hands should be washed each time the soiled mask is touched.

Acknowledgements.—We would like to thank the one hundred and twenty-nine doctors, nurses, students, and technicians who volunteered to help. We are also indebted to Messrs. Carus of Darwen for a supply of paper masks, to Dr. R. E. O. Williams for phage typing, to Mr. M. P. Curwen for help with statistics, to Miss Olive Duke for technical assistance, and to the Medical Research Council for a grant.

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THE LATE RESULTS OF HELLER'S OPERATION FOR CARDIOSPASM

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INTRODUCTION

Since Thomas Willis first described cardiospasm in 1672, its treatment has been empirical, for in spite of the many theories offered for its causation we still remain completely ignorant of its aetiology. Of the many types of treatment, dilatation with the mercury bougie, dilatation with a hydrostatic bag, and cardiomyotomy are the only methods of value; and by far the best of these is cardiomyotomy, first done by Heller in 1913. Even this operation has disadvantages, for it may lead to reflux œsophagitis; only a critical assessment of the results of operation will reveal that such patients have reflux, for most of them have been so delighted to be relieved of dysphagia that a minor degree of heartburn is no cause for complaint. But when we first noted that such symptoms did sometimes occur after Heller's cardiomyotomy, we studied critically a group of 42 patients, all suffering from cardiospasm, 28 of whom had a Heller operation. The results of this study we now present.

MATERIAL

From 1948–56 inclusive 42 cases of cardiospasm were admitted to the thoracic unit of the Manchester Royal Infirmary; 41 of these were treated, and all except one of these patients have been followed up in the past eighteen months. This has been done by clinical examination and barium-meal examinations in all; œsophagoscopy has been done in only a few cases.

CLINICAL FEATURES

The average age of the patients was 43 years, the youngest being 21 years and the eldest 65. There was no sex predominance, the cases being equally divided between males and females. This is contrary to the general belief that in cases of cardiospasm females predominate over males. The length of history varied from one to twenty years, the average being between three and four years.

Dysphagia, intermittent at first but becoming steadily worse, was a constant symptom. In some patients there was retrosternal or epigastric discomfort, sometimes radiating to the interscapular region and usually coinciding with the periods of dysphagia. In a few cases weight-loss was extreme, especially in those with long histories.

Malnutrition may be so severe that avitaminosis occurs; we have seen patients with peripheral neuritis from this cause. In such cases the cardiospasm is of such long duration that the patient has become accustomed to dysphagia and no longer complains about it; it is the neuropathy which brings him to hospital. The same may happen in the case of respiratory complications of cardiospasm. We have seen patients who complained of bronchitis due to aspiration of unswallowed œsophageal contents; they had become so accustomed to dysphagia that they did not regard it as abnormal, but sought treatment for the lung trouble.

Two features particularly need mentioning as we feel that neither is sufficiently emphasized in most reviews of cardiospasm.

1. Pain.—Cardiospasm is regarded by many as a painless condition. In cases where severe pain occurs another cause is usually sought. However, though gastric ulcer, coronary disease, and gallstones must be excluded, it must be realized that severe pain may be due to cardiospasm itself. A history of pain was obtained in 23 (57·5 per cent) of our cases. The commonest situation of pain was retrosternal or epigastric, occurring when swallowing was attempted. This pain either remained localized or radiated to the left hypochondrium, interscapular region, or up into the neck. This pain on swallowing occurred in 17 patients; in none of these was there a peptic ulcer. In 6 cases, however, spasmodic epigastric pain occurred which had no specific relation to the act of swallowing and usually came on between half an hour and two hours after taking food. In 5 of these cases, a peptic ulcer of the stomach was found. The diagnosis was suspected in the sixth case, but no proof was obtained.

It was Sir James Walton (1925) who divided cardiospasm into two clinical types, the painful and the painless. It is probable that spasm may be the cause of the pain, for œsophagitis and peptic ulceration cannot be demonstrated in all these cases.

2. The Emotional Factor.—Though there is no evidence that patients with cardiospasm are neurotic, in some its onset can be traced to some emotional disturbance.

Table I shows that such a factor was present in 16 of our cases.

Table I.—Emotional Factor in Cardiospasm

Emotional Factor	*No. of Cases*
Pregnancy	5
Attempted suicide	1
Street accident	3
Change of occupation	1
Death of a relative	2
Nursed a man who later committed suicide	1
Nervous breakdown	2
Commencement of military service	1

Apart from these specific instances, many patients stated that some emotional upset would precipitate acute dysphagia. Thus there seems to be strong evidence that the onset and aggravation of symptoms of cardiospasm are related to disturbances of the nervous system.

DIFFERENTIAL DIAGNOSIS

The radiological features of cardiospasm may be simulated by a carcinoma of the cardiac end of the stomach, and long-standing cases of cardiospasm may develop a malignant growth at the cardia.

The radiographs shown in *Fig.* 263 demonstrate two such cases. *Fig.* 263 A is a barium swallow of a

patient who presented with a recent history of progressive dysphagia. The radiograph indicated cardiospasm associated with a diverticulum of the lower end of the œsophagus. On œsophagoscopy there was a hard stricture, and at operation a carcinoma at the cardiac end of the stomach was found. *Fig.* 263 B is a barium swallow of a patient who had

A B

FIG. 263.—A, Carcinoma of the cardia simulating cardiospasm. An œsophageal diverticulum is shown as an incidental finding. B, Carcinoma of the lower third of the œsophagus affected by cardiospasm.

had cardiospasm for twenty years, but for six months the dysphagia had been worse, with pain and loss of weight. At œsophagoscopy carcinoma was found. This was confirmed at operation and, indeed, the growth was filling the lumen of the dilated œsophagus and had eroded through the wall of the œsophagus to involve the right lower lobe. We have seen two such cases.

A number of similar cases has been reported in the literature, but these two cases illustrate two important points. First, the radiological diagnosis of cardiospasm should not always be accepted without confirmation by œsophagoscopy. Second, a carcinoma may develop in an œsophagus affected by cardiospasm, but owing to the wide œsophageal lumen it may not produce noticeable symptoms until it has grown to a large size.

TREATMENT

Table II shows how our cases were treated. In the untreated case operation was considered, but the patient was unfit and he subsequently died. In such bad-risk cases we have done preliminary gastrostomy on three occasions; the definitive operation (Heller's) for cardiospasm was postponed for one to two months until the patients were in a better state of nutrition.

Octyl nitrite is prescribed by us particularly for early cases of cardiospasm because sometimes it may be effective in relieving attacks of dysphagia. However, it is rarely of lasting benefit, though we have one patient who has been using an octyl nitrite inhaler for four years and has required no other treatment.

The Negus hydrostatic dilator has only been used in two of our cases since 1948. In the years

Table II.—TREATMENT IN 42 CASES OF CARDIOSPASM

Heller's operation	28
Self bouginage	6
Œsophagoscopy and dilatation	5
Octyl nitrite	1
Intubation	1
No treatment	1

1946–7, however, it was used more often; but we were never satisfied that we had produced relief of dysphagia equal to that obtained by open operation. We achieved temporary improvement, but relapse was the rule. We may have been too timid in its use, but we were never satisfied that blowing up a balloon to stretch the cardia was really any safer than skilful digital dilatation by the method of Mikulicz; and though we had seen some successes from the latter method, we knew of some unfortunate complications. Both methods were blind, and we felt that the safest method for us was an open operation.

Other methods of treatment listed in the table scarcely warrant special comment, for the numbers are small and the methods have largely fallen into disrepute. Those patients treated by bouginage with a mercury tube had used the method for years and were disinclined to undergo a major operation.

Cardiomyotomy.—Twenty-eight cases were treated by Heller's operation. The operation was done through a laparotomy incision in 5 cases and a thoracotomy incision in 23 cases. It followed the standard procedure, and the myotomy extended over an average length of 7·5 cm.—5 cm. above and 2·5 cm. below the cardio-œsophageal junction.

Results.—The results are classified in *Table III* (assessed two to seven years after operation).

Table III.—RESULTS OF HELLER'S OPERATION FOR CARDIOSPASM (28 CASES)

	Laparotomy	*Thoracotomy*	*Total*
Good	1	10	11
Satisfactory	0	8	8
Failed	4	4	8
Untraced	0	1	1
Total	5	23	28

Good: These 11 cases were entirely symptom-free and were able to eat normal meals.

Satisfactory: These 8 cases were much improved by the operation, but they had mild symptoms such as occasional dysphagia or heartburn, insufficient to require further treatment.

Failed: Classified under this heading are 8 patients who developed either recurrent cardiospasm or complications attributed to the operation, severe enough to warrant further treatment.

Untraced: One case.

Six out of the 8 failures developed recurrent cardiospasm, 3 cases after a period of between 8 and 18 months, 2 after 3 years, and 1 after 6 years. Two of these patients have recently undergone a further operation for recurrent cardiospasm. For the remaining 4 cases which developed recurrent cardiospasm, it is probable that Heller's operation will have to be repeated.

The remaining 2 cases developed œsophageal strictures attributable to severe reflux œsophagitis. One patient who developed an œsophageal stricture was treated by a cardioplasty. A further stricture

A B

FIG. 264.—A, Case of cardiospasm before Heller's operation. B, Same case after Heller's operation. A hiatus hernia is shown.

developed later and he was subsequently treated by self-bouginage. The other stricture is being treated by self-bouginage.

Complications.—There was no mortality and no significant morbidity in the immediate post-operative period, but when these patients were later interviewed for the purpose of this investigation, 14 out of the 28 patients treated by Heller's operation complained of heartburn. In 5 it was mild and unassociated with other symptoms. In 9 it was severe and associated with regurgitation of food or fluid. The symptoms were worse at night in bed or when stooping or bending. All the patients had barium-meal examinations, but a hiatus hernia was found in only 3, though in 4 others the symptoms were so typical that a clinical diagnosis of incompetent cardia was made. Two patients developed œsophageal strictures and have already been mentioned. Both these cases and the three with proven hiatus hernia have undergone œsophagoscopy. In the two stricture cases and in one hiatus hernia, definite evidence of reflux œsophagitis was found.

Two of the cases with hiatus hernia have been operated on and the hernia has been repaired (*Fig.* 264). Both these cases had recurrent cardiospasm as well, though it is not suggested that there is necessarily any connexion between the two conditions.

DISCUSSION

Two main points for discussion have arisen out of this clinical investigation. First, a proportion of cases of cardiospasm recur after Heller's operation. Second, reflux œsophagitis is a complication of Heller's operation. Can its occurrence be prevented?

Recurrence of Cardiospasm.—It should be noted that recurrence was delayed for three years in two of our cases and six years in one. This indicates that a long-term follow-up is necessary before results of this operation can be judged.

There is no established cause for recurrence. When Heller first did the operation in 1913, cardiospasm was thought to be related to active spasm of the circular muscle-fibres at the lower end of the œsophagus. Heller found that a high proportion of his early cases recurred and felt that this was due to bridging of the muscle incision by scar tissue. This fixation of the circular muscle-fibres restores its sphincter action. To combat this Heller made two incisions, one anterior and one posterior, through the muscle of the œsophagus. Though we do not suggest that two incisions are necessary, there is no doubt that recurrences can be partly minimized by making sure that all muscle-fibres are divided down to mucosa and that a reasonable area of mucosa is exposed.

Another factor which may be important is the length of the myotomy, for the longer the myotomy on the œsophageal side, the better the result. Undoubtedly it is easiest to do a long myotomy on the œsophagus through a thoracotomy incision, and more difficult to do it by laparotomy. This may be why four of our five patients who had an abdominal approach were not completely relieved of their cardiospasm.

In the two cases which were operated on for the second time scarring across the divided muscle-fibres was noticeable. Having gently divided through the scar tissue, the myotomy was then extended proximally as far as the level of the inferior pulmonary vein in an endeavour to effect a more permanent result.

Reflux Œsophagitis.—A recent article by Acheson and Hadley (1958) has renewed interest in this problem. As they state, reflux œsophagitis is a complication of operations which interfere with the mechanism of the cardio-œsophageal junction, such as œsophago-gastrostomy. It has not, however, been widely recognized after Heller's operation. Acheson and Hadley imply that its occurrence may be an indication of the success of the operation. We would not agree with this. We feel that this complication should be and can be avoided.

When the operation is done through the chest, the cardiac end of the stomach is delivered through the hiatus in order to do the myotomy. This inevitably creates a temporary hernia. At the end of the myotomy the stomach is replaced below the diaphragm and the wound closed. No formal attempt

is made to repair the hernia produced by the surgeon, so that it is left to chance or post-operative adhesions to prevent symptoms of incompetence of the cardia or recurrence of a sliding hernia. If the myotomy is done through a thoracotomy, then we think that the Heller operation should be followed by a repair of the hernia which has been produced. It is not enough to be content with pushing the stomach down and taking no steps to prevent it prolapsing back into the chest. It may be that the abdominal route will prove to cause this complication less frequently; but the poor results in 4 of our 5 cases have not encouraged us to adopt the abdominal approach. *Fig.* 265 illustrates our technique of operation.

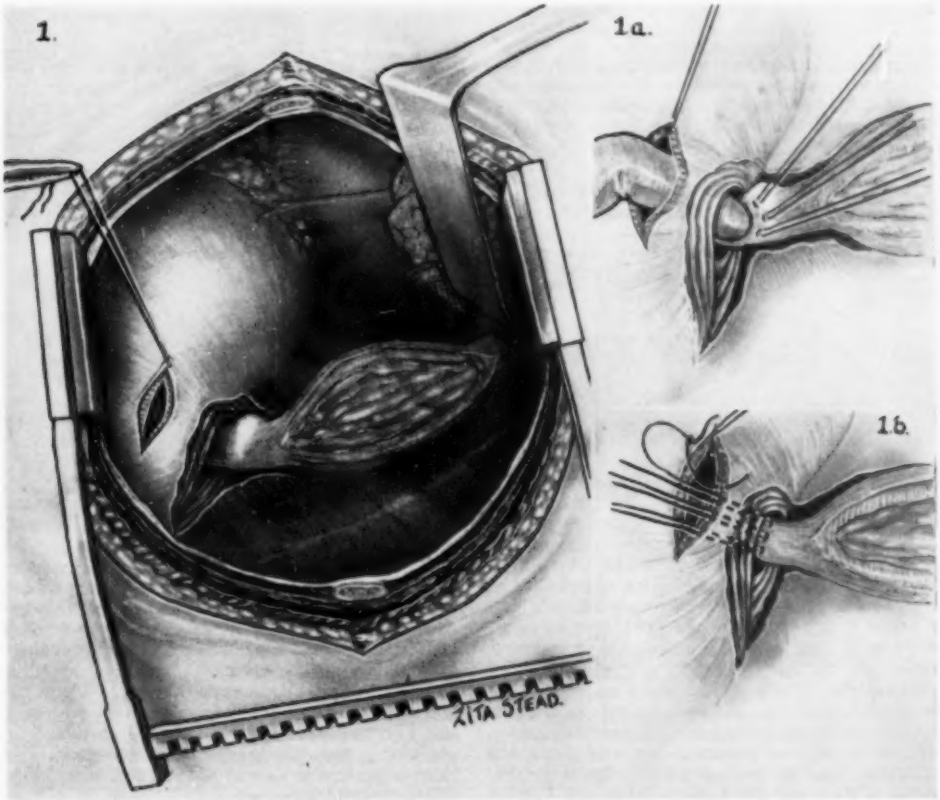

FIG. 265.—Heller's operation with additional technique for repairing hiatus hernia produced. **1**, Myotomy completed. Diaphragm opened prior to repair of hernia. **1 a**, Three sutures inserted through hernial sac. **1 b**, Hernia reduced by passing sutures under diaphragmatic hiatus; these sutures are tied on under surface of diaphragm.

SUMMARY

Forty-two cases of cardiospasm are reviewed. In 28 cases a Heller's operation was done, and 11 of these gave excellent results. The causes of failure are discussed. Reflux œsophagitis may complicate an otherwise excellent result. An operative technique is discussed which aims at preventing this complication.

Acknowledgements.—It is a pleasure to acknowledge our indebtedness to our old colleague, Mr. Graham Bryce, who has allowed us to include some of his cases in this series. We thank the Department of Medical Illustration at the Royal Infirmary, Manchester, for their help in the preparation of the illustrations.

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TRAUMATIC HÆMOBILIA WITH SEVERE HÆMATEMESIS AND MELÆNA

REPORT OF A CASE AND REVIEW OF THE LITERATURE

By B. R. SWORN, Stafford

The presence of blood in the bile-ducts may be due to any one of a number of lesions. It may occur with trauma or disease of the liver, gall-bladder, or bile-ducts, injuries of the vessels, either the hepatic or cystic arteries or the portal vein, or as a reflux from the duodenum. In most of these cases it is relatively unimportant and has received little surgical notice.

Sandblom in 1948 collected a number of cases of what he called "traumatic hæmobilia" in which the bleeding was from a liver injury and might be sufficient to produce hæmatemesis and melæna. The characteristic features he described were a sub-capsular tear of the liver from external injury, a latent period of days, weeks, or even months followed by attacks of pain suggestive of biliary colic, and the presence of blood in the bowel.

He reported one case of his own and was able to trace eight others in the literature which more or less satisfied the criteria. The word 'hæmobilia' was chosen because of the similarity to hæmaturia from rupture of the kidney.

His first case had been reported by Owen in the *London Medical Gazette* of 1848 and appears to be the only one recorded in this country. The patient was a young gentleman of 22 years who was thrown from a light chaise while racing with a companion on horseback. He seems to have got up himself, but when asked if he was hurt answered "he was afraid he was a good deal hurt internally". Seen a couple of hours later he was in a state of profound shock with no external injuries. The shock improved, but on the second day he complained of severe pain in the right upper abdomen passing up to the right shoulder. On the third day the pain was less and there was faint jaundice, and on the fourth day he had a number of attacks of pain and the jaundice increased, but the abdominal pain was less. On the fifth day there was frequent passage of bright blood per rectum and it was noted that griping abdominal pain preceded each motion. After this he had melæna, in considerable amounts, which persisted for some days. He died on the twelfth day, apparently from blood-loss with suppression of urine and abdominal distension.

At autopsy, the liver was much enlarged and there was a deep laceration closed off by recent adhesions to the diaphragm. Apart from congestion there was no lesion of the bowel to cause bleeding. Owen was unable to trace any connexion between the liver cavity and the bowel; as Sandblom remarks, it is probable that the communication that he missed was the bile-duct.

Of the 9 cases reported, obvious bleeding into the gut occurred in 5, occult blood was found in 1—Sandblom's own patient—and in the other 3 it was not mentioned in the notes; 2 patients died, both from bleeding.

Sparkman, in 1953, confining himself to traumatic hæmobilia with massive blood-loss, reported 1 case and found 13 others in the literature. He agreed with the diagnostic criteria, i.e., a non-penetrating abdominal injury followed, after a latent period, by pain suggestive of biliary colic and bleeding into the bowel, but did not accept the importance of the original liver injury being subcapsular. He found, in fact, that in a majority of cases there was a laceration of the liver which had either been treated surgically (4 cases) or had been, at least partly, closed naturally by adhesions to the diaphragm. Sparkman, therefore, considers that deep lacerations of the liver should not only be cleared of traumatized tissue as far as possible, but that a drain should be put down to the depth of the wound and the tissues closed round it to minimize the risk of a deep cavity forming, a procedure advocated by Krieg (1936). Sparkman's own case of hæmobilia followed the primary suture of a deep tear.

In this series there was a mortality of 50 per cent. Of the 7 patients who died 4 had no operation and death occurred 11 days, 17 days, 3 months, and 3–4 months respectively, after injury. In 2 cases the initial laceration had been treated either by packing or suture, and in 1 other case drainage of the common duct was carried out, but the bleeding continued and the patient died two weeks later.

Of the 7 who survived, 2 had a cholecystectomy, and in one of these a biliary fistula formed soon after the operation. Two had deliberate drainage of the gall-bladder and 1 drainage of the common duct. In 2 cases, both of which had had primary suture of a laceration, the deep cavity was later opened up through the scar and packed, one of them having had a previous drainage of the common duct without effect on the bleeding.

Barling in 1956 reported a further case of massive hæmobilia and reviewed the literature with regard to diagnosis and treatment.

His patient was a boy of 17 years who had been shooting kangaroos from a motor-cycle and was thrown across the handle-bars when he rode over a tree stump. The next day he was operated on for a ruptured liver, which was closed by deep mattress sutures. Some two weeks later he had jaundice, severe abdominal pain, and a hæmatemesis. After three similar attacks he was re-operated on with the diagnosis of adhesive biliary obstruction, but as the liver appeared normal except for omentum adherent to the scar and the common duct was not dilated, nothing was done. Three weeks later the jaundice, pain, and bleeding recurred. He was again re-operated on but, apart from multiple adhesions, no lesion was found. He continued with the attacks of jaundice, pain, and bleeding for some weeks, but eventually they subsided and he recovered.

Barling stresses that traumatic hæmobilia is not generally recognized as a possible cause of hæmatemesis and melæna. As a result, multiple laparotomies are often done before the diagnosis is made. He does not consider that simple drainage is likely to be effective and advocates either a direct attack on the cavity or partial hepatectomy.

CASE REPORT

L. W., a farm labourer aged 34 years, was involved in a motor-cycle accident on Feb. 1, 1958. He was admitted to hospital with slight concussion, fracture of left wrist, and injuries to the left leg and abdomen. The fracture was reduced under general anæsthesia and fixed in plaster. On Feb. 4 he was discharged with no further note on his abdominal injury. He was re-admitted on Feb. 26 with

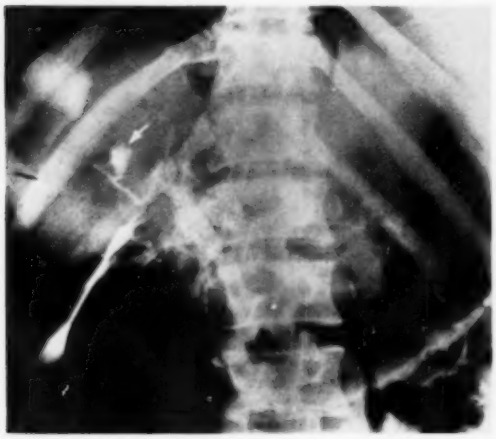

FIG. 266.—Hepatic angiogram. Arrow points to defect in right hepatic artery.

hæmatemesis and melæna. Since his accident some three weeks before he had complained of intermittent abdominal pain. For some days there had been very dark and frequent motions and in the past two days he had vomited a number of times, either dark 'coffee-ground' material or obvious blood, with more persistent pain in the epigastrium and right hypochondrium.

ON EXAMINATION.—He was pale and complained of pain and tenderness in the epigastrium and right hypochondrium. There was some blood on rectal examination. The pulse-rate was 120, blood-pressure 90/50, hæmoglobin 59 per cent. The pain and bleeding recurred at intervals and on March 13 he became jaundiced. He had had 7 pints of blood, and hæmoglobin was now 44 per cent, but the serological tests suggested that the jaundice was obstructive. The jaundice subsided in 3–4 days and did not recur, but the bleeding persisted.

On March 18, after another severe hæmatemesis, it was apparent that the attacks of pain immediately preceded the bleedings. The patient's general condition was poor, the hæmoglobin had been 38 per cent the day before, and there was a tender swelling below the right ribs (? enlarged liver). After further transfusions the hæmoglobin was 50 per cent and an exploratory operation was done.

AT OPERATION.—The stomach and duodenum appeared normal. The tumour was an immensely distended gall-bladder, plum-coloured, and aspiration confirmed that it contained a large quantity of dark fluid blood. The liver appeared quite normal in size and consistency and there were no adhesions or other signs of injury. The common duct was not dilated and contained normal bile. The gall-bladder was removed and also a piece of liver for biopsy. The excised gall-bladder showed no obvious source of the bleeding, so the common duct was drained after being opened and explored without result. At the end of the operation normal bile was draining away, but half an hour later there was free bleeding from the tube. The blood was bright red, with no clots or mixture of bile, and clotted well in the container. The quantity was about 6 oz. During the ten days the drain remained in the duct there were several more bleedings. Each time the patient complained of severe pain shortly before the blood appeared and there were no clots in it.

PATHOLOGICAL REPORT (Dr. F. Pick).—"The gall-bladder wall is greatly thickened. The epithelium is mostly atrophic, with areas of ulceration and hypertrophy. The submucosa and serosa show marked œdema, multiple hæmorrhages, and chronic inflammatory infiltration. The liver tissue shows marked congestion, chronic inflammatory infiltration of periportal spaces, and bile-duct proliferation. The picture is that of chronic cholecystitis

FIG. 267.—Liver, showing dilated bile-duct with large recent clot.

with superimposed acute cholecystitis and chronic inflammatory changes in the liver."

Because of the liver changes, a full blood-examination with clotting and bleeding times was done, but showed no abnormalities except those of a secondary anæmia.

On April 5 the patient first complained of severe pain in the back and this remained a constant feature. A splenic injection with contrast medium (Dr. MacDonald Holmes) showed a normal portal system, but a biligrafin X ray failed to outline the ducts.

On April 10, the attacks of bleeding with abdominal and back pain persisting, a second exploratory operation was done with the object of cholangiography and hepatic arteriography. Again, there was no evidence of any lesion of the liver and the ducts and vessels were isolated and examined as far as possible into the portal fissure. The common duct was now found to be dilated, but contained normal bile and the contrast injection showed a normal duct pattern. The patient's condition deteriorated and the arteriogram had to be abandoned.

The following day he collapsed after bringing up a quantity of bile-stained blood and a very 'bubbling' chest. From this time we were unable to raise the hæmoglobin above 20 per cent in spite of transfusions (the patient had more than 40 pints in all) and he died on June 28, five months after the original injury.

POST-MORTEM.—The hepatic artery was injected and radiograph showed a leak from one of the main right branches into the centre of the liver (*Fig.* 266). After removal of the liver, which was of normal size, a small linear scar was seen on the posterosuperior surface of the right lobe and deep to it was a central cavity, 3 cm. in diameter, almost filled with laminated clot. The cavity opened into a grossly dilated bile-duct containing a large adherent clot which must have formed after the cholangiogram had been done (*Fig.* 267). Nothing else of significance was found.

DISCUSSION

The case reported here carries on the somewhat melancholy story of missed diagnosis due to lack of knowledge. The consensus of opinion among those writing on the subject is that early diagnosis and the avoidance of unnecessary operations depend on a knowledge of the condition, and that this is unlikely to be sufficiently widespread until traumatic Hæmobilia finds a place in the text-books.

The importance of the rather trivial abdominal injury was not fully appreciated and because of this, and the biopsy, the possibility of liver disease was considered rather than trauma as the primary factor. The first reference found to what appeared to be a somewhat similar case was a recent paper "Hæmocholecyst following ruptured Aneurysm of Portal Vein" (Barzilai and Kleckner, 1956) in which a young woman with chronic liver disease died from severe intestinal bleeding. It was only later that the relevant literature was traced.

As well as the triad of liver injury, biliary-type colic, and blood in the bowel, jaundice and a hæmocholecyst have been present in 50 per cent of the recorded cases, although not always in the same patient.

The pain which is such a distinctive feature has been ascribed first to the increased tension produced by bleeding into the liver cavity and then to the passage of clots down the biliary tracts. There is no doubt that blood may clot in the presence of bile, but an increase of pressure in the bile-ducts is sufficient to produce severe biliary colic, as demonstrated by McGowan, Butsch, and Walters (1936), and so typical biliary colic may occur with bleeding even though there are no clots.

Treatment.—Since all the deaths in the recorded cases of traumatic hæmobilia have been due to loss of blood, the main aim of treatment must be to stop the bleeding, but according to Sparkman (1953) prophylactic treatment is also important. In his view deep lacerations of the liver should be drained and not completely closed, so as to prevent the formation of a deep cavity. Of the total of 21 cases, 5 had had immediate suture of a laceration, but in 2 others, in which the details had been personally reported to Sparkman, the laceration had been either packed or drained.

In the 16 cases with massive bleeding the mortality was 50 per cent. Drainage of the bile-ducts, although frequently carried out, does not appear to have any great value in the treatment. In only 2 cases, both of which recovered, was any direct attack made on the source of the bleeding. In these two, which had followed the primary suture of a laceration, the cavity was opened up through the old scar and packed. This may well be the best operation under such circumstances, but to locate and pack a central cavity through normal liver tissue is likely to be a hazardous procedure.

If there are no surface indications hepatic angiography would seem to be the best method of confirming the diagnosis and localizing the lesion.

As already mentioned, Barling (1956) suggests that partial hepatectomy should be considered for some patients. Another possibility is ligature of the hepatic artery. In the past this was commonly followed by early necrosis of the liver, but Markowitz, Rappaport, and Scott (1949) found, during experimental work on dogs, that penicillin prevented this necrosis which was presumably due to organisms in the portal blood. Hepatic ligature has now been fairly extensively tried out in the treatment of cirrhosis of the liver, and although it has not met with much success for this, it has been shown that ligature of the vessel proximal to its gastro-duodenal branch and under cover of penicillin and streptomycin is a reasonably safe procedure.

SUMMARY

Traumatic hæmobilia was first named and described as an entity by Sandblom in 1948. The clinical features are a non-penetrating abdominal injury, a latent period of days, weeks, or even months, followed by abdominal pain, biliary colic, and the presence of blood in the bowel.

It is a rare condition, 21 cases in all having been reported, but 16 of these have had severe hæmatemesis and melæna with a mortality of 50 per cent.

The correct diagnosis has seldom been arrived at until a late stage, and only 2 cases have had radical surgery, by opening up and packing the central liver cavity.

In view of the high mortality from bleeding, either partial hepatectomy or ligature of the hepatic artery may be justified in some cases.

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TRANSTHORACIC AORTIC CONTROL IN THE MANAGEMENT OF ACUTE RUPTURE OF AN ABDOMINAL AORTIC ANEURYSM

By C. R. SAVAGE, Warwick

More than 30 cases of ruptured abdominal aortic aneurysm treated by surgical excision and grafting have now been reported (Bahnson, 1954; Cooley and De Bakey, 1954; Gerbode, 1954; Javid, Dye, Grove, and Julian, 1955; Shumaker and King, 1955; Rob, 1955; Farrar, Adson, Kirklin, Martin, and Barloh, 1956; Milnes Walker, 1957; Barrett-Boyes, 1957; Jamplis, 1958; Cockett, 1958). Different degrees of severity of rupture may occur. In the most marked death may follow in a few minutes, while in the mildest there is progressive slow leakage of blood with chronic hæmatoma formation. The surgical management of this latter group differs little from that of uncomplicated aneurysms, but dissection and control of the aorta above the aneurysm are likely to be more difficult. It is, however, in the intermediate type of case, where the retroperitoneal tissues and sometimes the peritoneal cavity itself are filled with blood and blood-clot and shock is marked, that proximal control of the aorta is most urgently required at an early stage. In its absence sudden severe and even fatal hæmorrhage may occur during dissection of the aneurysmal sac or of the aorta immediately proximal to it. This segment of the aorta below the renal arteries is sometimes very short and its walls may be thin and friable. There are two main methods of control available. Compression of the aorta against the vertebral column can be carried out above the level of the renal vessels. Owing to the interposition of soft structures, in particular the pancreas, this may be difficult to maintain effectively and visceral damage may also result. More adequate control may be obtained by isolation of the lower thoracic aorta through an extension of the abdominal incision or through a separate thoracic incision. Further possible advantages of this method carried out as a preliminary procedure have been suggested by Merendino (1956). It may be of assistance in the management of the hypotension which can occur during anæsthetic induction from suppression of vasoconstrictor mechanisms, or limit further hæmorrhage that might occur following the relief of any tamponade effect of raised intra-abdominal pressure by laparotomy.

Fig. 268.—Radiograph of the abdomen showing calcification in the wall of the aneurysm.

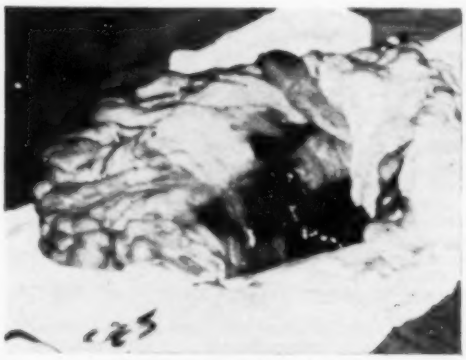

Fig. 269.—Photograph showing the retroperitoneal hæmatoma.

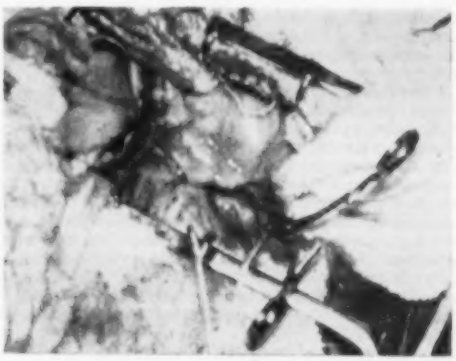

Fig. 270.—Photograph showing, at the upper end of the thoracic incision, the tape and arterial occlusion clamp in position on the thoracic aorta.

The following case of acute rupture of an abdominal aortic aneurysm is reported to illustrate the value of transthoracic aortic control during the

dissection of the abdominal aorta and the aneurysmal sac.

CASE REPORT

A man aged 70 years was admitted on April 2, 1958 complaining of the sudden onset of severe upper abdominal pain radiating through to the back, of two hours' duration. He had vomited twice and his bowels had acted normally earlier the same day. He had had previous mild attacks of pain in the same region intermittently for the past year. Three years previously a diagnosis of abdominal aortic aneurysm had been made at another hospital. Surgical treatment had been advised but not carried out. Radiographic examination at that time had shown calcification in the wall of the aneurysm (*Fig.* 268).

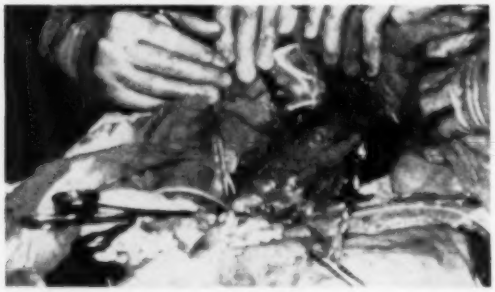

FIG. 271.—Photograph showing the aortic aneurysm with dissection nearly completed. The occlusion clamp on the abdominal aorta is seen on the left side of the picture.

ON EXAMINATION.—The patient was severely shocked, pale, anxious, restless, thirsty, and in great pain and distress. Temperature was 97° F., pulse-rate 130, respiration 24, blood-pressure 100/70. There was marked upper abdominal tenderness and guarding, and a large pulsating swelling was felt in the epigastrium and umbilical region. Femoral pulsation was present on both sides. A diagnosis of ruptured aortic aneurysm was made. After treatment of shock and blood replacement, operation was performed two hours after admission.

AT OPERATION.—The peritoneal cavity was opened through a long left paramedian incision. There was a small quantity of blood present and on displacing the intestines a large retroperitoneal hæmatoma was seen overlying an aortic aneurysm and extending widely into the surrounding tissues (*Fig.* 269). Before beginning dissection of the sac the abdominal incision was extended into the 8th intercostal space and the left pleural cavity opened. The lower thoracic aorta was then dissected free for about 5 cm. and a tape and occlusion clamp placed in position for use in the event of sudden hæmorrhage from the aneurysm (*Fig.* 270). The aneurysm, which extended from 1 cm. below the renal arteries down to and including the aortic bifurcation, was then freed from the surrounding hæmatoma and the common iliac arteries were controlled (*Fig.* 271). During the dissection of the upper part of the sac there was sudden severe hæmorrhage from two linear splits on the anterior surface which was promptly controlled by closure of the clamp on the thoracic aorta. Rapid dissection behind the aneurysm was then possible and a further clamp was applied immediately distal to the renal arteries. The thoracic clamp was then released. The aneurysmal sac was next excised and replaced by an Orlon bifurcation graft (*Fig.* 272). Owing to gross disruption of the retroperitoneal tissues by clot, closure of the peritoneum over the graft was not possible.

Convalescence from operation was satisfactory, except for the development of an area of infected collapse in the upper lobe of the left lung which became cavitated, but resolved following antibiotic therapy.

FIG. 272.—Photograph showing the Orlon bifurcation graft in place following release of the occlusion clamps.

At the patient's last attendance four months later he was symptom-free, and the satisfactory circulation in his legs had been well maintained.

SUMMARY

A case of acute rupture of an abdominal aortic aneurysm treated by excision and grafting is reported. The value of control of the thoracic aorta before dissection of the aneurysmal sac is emphasized.

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GALL-STONE OBSTRUCTION OF THE SIGMOID COLON WITH PARTICULAR REFERENCE TO AETIOLOGY

By J. H. PRYOR, Worthing

Gall-stone obstruction of the intestine, although not common, is not infrequently the cause of an obscure intestinal obstruction in obese elderly females. Particularly is this so if the obstruction is intermittent. The site of the obstruction is most frequently the lower ileum 1–2 ft. from the ileocæcal valve, which is the narrowest part of the ileum. More rarely the stone is impacted in the sigmoid colon. The reason for this latter site is puzzling, as it would seem likely that any stone negotiating the ileum would readily traverse the colon and rectum. If, however, the fistula is between the gall-bladder and colon this obviously explains the stone being in the colon, but it does not explain why the obstruction is in the sigmoid. A case of gall-stone obstruction of the sigmoid is presented, which it is felt may shed light on this problem.

CASE REPORT

Mrs. E. M. B., aged 74 years, was admitted as an emergency to Worthing Hospital on Nov. 30, 1957. For two weeks previous to admission she had noticed intermittent colicky abdominal pain, during which there were episodes of retching, but no vomiting. She had some bowel actions during these two weeks, but frequently she felt there was a block. There was occasional passage of flatus, but a feeling "that there is more to come".

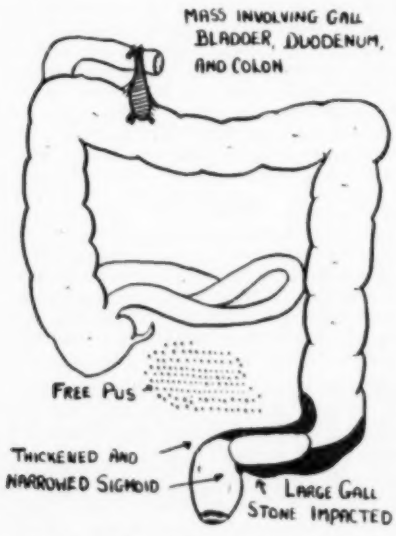

Fig. 273.—Schematic diagram of operation findings.

She had previously been in excellent general health and was a very active woman who regularly walked 7 miles daily. She denied any previous dyspeptic or bowel symptoms.

On Examination.—She was an obese, flushed, elderly woman with a distended abdomen. Temperature was 100° F., pulse-rate 98. The abdomen was very distended and hyperresonant. There was marked tenderness and guarding in the left iliac fossa, no mass being palpable. Bowel-sounds were frequent and active. The rectum was ballooned and empty. No mass could be felt in the pelvis. A provisional diagnosis of subacute intestinal obstruction due to diverticulitis was made and an emergency operation was performed after intravenous resuscitation.

At Operation.—At laparotomy through a right lower paramedian incision (*Fig.* 273) there was free seropurulent fluid. The cæcum was distended and the large bowel markedly dilated, leading to an obstruction at the middle part of the sigmoid colon. On inspection this appeared to be due to a hard foreign body occluding the lumen, probably a gall-stone. Proximal to this obstruction the whole bowel wall was thickened, inflamed, and bound by adhesions to the side wall of the pelvis. Distally, the bowel was similarly affected to the rectosigmoid junction. A hand passed up in the region of the gall-bladder revealed a mass involving the gall-bladder and colon; no stones could be felt in this mass. Diagnosis was made of gall-stone obstruction of the sigmoid. The cause of the inflamed colon was thought to be diverticulitis or possibly irritation from the impacted gall-stone.

Fig. 274.—Gall-stone removed from sigmoid colon.

The cæcum was aspirated to collapse the bowel. The stone was removed via a longitudinal incision in the colon, the segment of colon being isolated by light intestinal clamps as it was impossible to move the stone in either direction because of the narrowed sigmoid. The stone was a large non-faceted gall-stone measuring 1½ × 1 × 1 in. (*Fig.* 274). The bowel wall was closed in two layers. Because the bowel wall was unhealthy and friable a transverse colostomy was performed.

Post-operatively, intravenous fluids were maintained with the addition of intravenous tetracyn given at the rate of 250 mg. 6-hourly. Intermittent gastric suction was maintained.

The patient made a good recovery. The colostomy functioned the following day and convalescence was uneventful. Subsequently a barium enema was performed which showed diverticulitis of the sigmoid colon in the region at which the gall-stone was obstructed (*Fig.* 275). There was no radiological evidence of a colonic fistula.

Five weeks after the original operation the colostomy was closed, and convalescence from this was uneventful.

The patient was discharged eight weeks from the day of admission. When reviewed on March 1, 1958, she was in good health and symptom-free.

DISCUSSION

Gall-stone obstruction of the colon is a rare condition. The first description was made by Courvoisier, in 1890, who found a case in a series of post-mortem studies, although gall-stone obstruction of the small bowel was described by Bartholini in 1654. Cases, usually single, have been reported in the literature from time to time.

FIG. 275.—Barium enema showing area of diverticulitis of sigmoid colon at site of obstruction of gall-stone.

The interesting feature in this case is the site of obstruction of the stone.

At operation it was uncertain whether the sigmoid colon wall was inflamed due to irritation from the gall-stone or due to diverticulitis, but the latter was shown to be the cause. It is very probable that it was the narrowing due to diverticulitis which caused the stone to obstruct at this site. When the stone was arrested it most probably became impacted by repeated peristaltic waves and secondarily by chemical and traumatic inflammatory reaction in the adjacent wall of the colon.

The question arises whether diverticulitis may be the exciting cause for obstruction of a gall-stone at the sigmoid colon in all or most cases. Symptomless diverticulitis is not infrequently found in obese elderly females, and there may well be a narrow segment of sigmoid due to this which is clinically silent. In this case, the diameter of the stone was only 1 in., and yet obstructed sigmoid colon, as seen in intestinal obstruction due to carcinoma of the rectum, may be up to three or four times this diameter. It is known that quite large gall-stones can pass naturally per rectum. Foss and Summers (1942) recorded a case in which a gall-stone 2½ in. in diameter was passed per rectum, thus relieving an acute intestinal obstruction, and so it is probable that obstruction in the sigmoid only occurs in the presence of diseased colon. Martin, in discussion of Foss and Summers's paper, quoted a case where obstruction occurred in a segment of sigmoid colon which was the site of a ruptured diverticulum which had been oversewn eight years previously. Harris, McNamara, and Dardinski (1947) recorded 2 cases of obstruction of the sigmoid forming fistulæ, but no indication of the cause of the obstruction was given. In Lloyd Williamson's case (1952) the bowel wall is described as very thickened, but the barium enema did not give a clear picture of diverticulitis. The author states that it was impossible to decide whether there was actual diverticulitis present.

Grey Turner (1932) described a case in which a gall-stone was found obstructing the transverse colon. The explanation he gave for the impaction in the transverse colon was spasm of the bowel wall, but he did not state the cause of the spasm.

Further review of the literature has been unrewarding on this point.

Regarding diagnosis, this case showed many of the classical features of gall-stone obstruction, but the extreme tenderness and guarding in the left iliac fossa pointed to a diagnosis of diverticulitis as the cause of the obstruction.

Regarding treatment, it is possible that recovery may have occurred without the performance of transverse colostomy, but it was considered the safest course to adopt.

SUMMARY

A case of gall-stone obstruction of the sigmoid colon is presented. Recovery followed removal of the stone and performance of transverse colostomy. The mechanism of obstruction at this site is discussed and it is suggested that there may frequently be a co-existing diverticulitis which is the exciting cause for the stone obstructing at this level.

Acknowledgements.—I would like to thank Mr. K. Whittle Martin, under whom this case was admitted, for his permission to operate and to record this case. I wish to thank Miss G. J. Awcock for the diagram and Mr. Higgins, Clinical Photographer, Royal Sussex County Hospital, Brighton, for the photographs.

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PARENTERAL FLUID THERAPY FOR CHILDREN UNDERGOING MAJOR ABDOMINAL SURGERY

A STUDY OF 120 CONSECUTIVE CASES

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THIS report describes a scheme for estimating the needs of infants and children who require intravenous infusions in support of major abdominal operations and the results of its application. Previous experience of episodes of severe clinical deterioration, later judged to have been due to defects in post-operative fluid therapy, provided the stimulus to this investigation.

A clearly defined plan of treatment is more important for infants and children than for adults for the following reasons:—

1. The wide variation in the body mass from about 3 lb. in newborn premature infants to 80 lb. in children aged 13 years.

2. Owing to difficulties in making an early diagnosis of acute inflammatory conditions of the abdomen and the high incidence of intestinal obstruction from congenital abnormalities of the bowel, children are frequently dehydrated at the time of an operation and may need intravenous therapy for days or even weeks afterwards. Fluid and electrolytes should therefore be given to supply the patient's needs as fully as possible from the outset. The restoration of deficiencies cannot be safely postponed until oral feeding is resumed, as has sometimes been recommended for potassium in adults (Black, 1953).

3. The renal function of newborn and very young infants has been shown to be less efficient than that of older subjects. Both glomerular filtration rate and tubular function are lower relative to body size, and the ability to concentrate and to dilute the urine is also less (McCance, 1948, 1950). Although renal function in full-term and even in premature infants matures soon after birth (Barnett, Hare, McNamara, and Hare, 1948; Rubin, Bruck, and Rapaport, 1949; Pratt, Bienvenu, and Whyte, 1948; McCance and Widdowson, 1954), it deteriorates more rapidly and to a greater extent than in older subjects if they become dehydrated (Calcagno and Rubin, 1951). Amounts of fluid which will relieve dehydration completely must therefore be given.

The fluid and electrolyte requirements are assessed according to body size and to the probable changes in the volume and composition of the body fluids.

Body-fluid Changes.—

Before Treatment in Dehydrated Patients.—Dehydration due primarily to lack of water is proportionately distributed between the extra- and intracellular volumes of fluid, and during the process potassium leaves the cells and is excreted in the urine (Elkinton and Taffel, 1942). In contrast, dehydration due to vomiting or to 'pooling' of gastro-intestinal secretions, which contain large amounts of sodium salts, mainly reduces the volume of the extracellular fluid, including the blood (McCance, 1936; Marriott, 1947). The dehydration associated with acute abdominal conditions is usually of mixed origin. Anorexia has reduced the intake of water, pyrexia has increased the output of water in the insensible losses from the skin and lungs, and both water and salt have been lost in the sweat and from the gastro-intestinal tract. High intestinal obstruction with vomiting and 'pooling' of gastro-intestinal secretions containing an excess of chloride relative to sodium leads to alkalosis with excretion of potassium in the urine. The alkalosis cannot then be corrected by giving sodium chloride without potassium (Darrow, 1945; Wallace, 1952). Infants have relatively larger losses from the skin and lungs and from the bowel than older subjects and consequently reach advanced stages of dehydration in a shorter time.

During the Course of Treatment.—Selective excretion of water is adversely affected by sodium depletion, and in adults by a post-operative antidiuretic effect (Le Quesne and Lewis, 1953), variously attributed to pre-operative anxiety, premedication or anæsthesia, and to the trauma itself (Le Quesne, 1957), but this antidiuretic effect has not been described in young children.

Solutions of sodium salts without potassium promote over-retention of sodium which expands the extracellular fluid volume, resulting sometimes in œdema, and replaces potassium in the cells, the potassium being lost in the urine (Stewart and Rourke, 1942; Gamble, Wallace, Eliel, Holliday, Cushman, Appleton, Shenberg, and Piotti, 1951). The extent of these changes is likely to be enhanced during the post-operative period, since the endocrine response to the stress of trauma and shock is to retain sodium and to excrete potassium. As treatment proceeds a progressive depletion of potassium superimposed on uncorrected pre-operative losses may lead to serious clinical manifestations with falling concentrations of potassium in the plasma and extracellular fluid. The early signs of this deficiency are not easy to discern in very sick patients and may be attributed to the abdominal condition itself. Since the serum potassium levels of normal newborn infants are often high, it has been supposed that they are unlikely to develop depletion, and that administration of potassium could be safely postponed. This is not true for those with intestinal obstruction who have become dehydrated and therefore depleted of potassium, for their bowel function only recovers slowly, and while ileus persists further losses increase the deficit. Tudvad, McNamara, and Barnett (1954) have shown that even premature infants given potassium intravenously are able to excrete it freely in the urine.

Aims of Fluid Therapy.—These are as follows: (1) To restore the circulating blood-volume and to establish and maintain a free flow of urine. (2) To restore the volume of the body fluids by administering a safe excess of water and sodium salts during the first twelve to twenty-four hours of treatment, and subsequently to maintain them by this means. (3) To

replace a deficit of potassium salts gradually and to avoid progressive depletion by giving potassium with the water and sodium salts. (4) To supply part of the caloric requirement and to spare protein, by giving dextrose solution from the outset, and protein (plasma) or protein derivatives (casein hydrolysate) to patients who are already depleted, or who may require the administration of the fluids for a long time. (5) To treat anæmia promptly by giving blood transfusions.

These principles have been widely applied to the treatment of infants with gastro-enteritis (Flett, Pratt, and Darrow, 1949) and patients in diabetic coma (Butler, 1950), where the clinical state is similar to that of surgical patients with acute abdominal conditions. Hesitation in applying them wholeheartedly to the treatment of the latter group of patients is due in part to the serious consequences of intravenous therapy observed in past decades, when the concentration of sodium salts in the solutions used was often excessive, causing gross peripheral, and sometimes pulmonary, œdema. Surgeons have therefore tended to restrict the volume of fluids by infusion to amounts far below those customarily given by the oral route, although these patients have often had severe gastro-intestinal losses both before and after operation. There is danger from giving too little as well as too much salt. For instance, the common practice of using 5 per cent dextrose in one-fifth isotonic saline solution will expose those who have sustained heavy alimentary losses to water intoxication, and if potassium is to be given systemically by infusion without risk, the volume of fluid prescribed must be sufficient to ensure a free flow of urine.

PLAN OF TREATMENT FOR INFANTS AND CHILDREN

The volume and composition of the solutions for infusion are varied according to the size of the patient and his clinical state. At the outset the severity of dehydration and electrolyte depletion is assessed from the history and clinical examination. It is convenient to prescribe separately the *initial infusion* for the first stage of treatment ending when urine is passed, and then *daily infusions* for 24-hour periods, adding extra amounts, *replacement infusions*, if abnormally high losses are sustained, as in paralytic ileus.

The composition of the solutions used is shown in *Table I*. They include (1) solutions of sodium chloride and lactate in concentrations suitable for infusion with 5 per cent dextrose, and as alternatives, diluted plasma or amigen; and (2) concentrated solutions of potassium chloride and acetate. The separate addition to the infusions of concentrated solutions of potassium salts is convenient and an effective reminder of the need for prescribing them with care.

Initial Infusion.—

Amount.—Manifest dehydration represents a loss of water of about 5 per cent of the body-weight, and therefore an infusion of 20–30 ml. per lb. (about 40–60 ml. per kg.) body-weight, given over a short period of 4–6 hours, is suitable for patients of all ages (*Table II*). Severely dehydrated infants, including newborns, are given the infusion at the higher rate (30 ml. per lb. in 4 hours). If urine is passed freely before the prescribed volume has been given a change is made to the daily infusion. If possible, the whole of the initial infusion should be given to a dehydrated patient before operation, the rate then being reduced to that of the daily infusion. The latter rate is also suitable at the beginning of an elective operation, but during a procedure which is likely to produce shock, e.g., exposure and handling of the bowel, or loss of blood, the rate of the infusion is increased at once.

Table I.—THE COMPOSITION OF SOLUTIONS FOR INFUSION* AND FOR ADDITION† TO INFUSIONS

*For Infusion**

Infusion Solution in 5 per cent Dextrose	mEq./l.			
	Na	K	Cl	HCO_3 Equivalent
Isotonic saline	154		154	
M/6 sodium lactate	166			166
⅓ normal saline	77		77	
⅙ normal saline	31		31	
⅓ strength plasma	70	2·5	50	15
Amigen 2½ per cent	31	6·4	22	

For Addition†

Concentrated Solution of Potassium Salt for Addition to Infusion	mEq./5 ml.		
	K	Cl	HCO_3 Equivalent
Potassium chloride 0·8 g.	11	11	
Potassium acetate 1 g.	10		10

* Further dilution of plasma to 1½ strength and amigen to 1½ per cent with 5 per cent dextrose (suitable for daily infusions) is conveniently achieved by using a calibrated mixing cylinder leading from a two-bottle infusion set.

† The addition of 10 mEq. potassium salt to each 500 ml. infusion solution raises the concentration of potassium by 20 mEq./l. This amount is suitable for addition to all the solutions listed, except amigen, to which a smaller amount should be added.

Composition.—Solutions of dextrose with sodium, but without potassium salts, are given at this stage, the concentration of sodium salts being chosen according to the clinical state of the patient. Thus one-fifth isotonic saline for patients before elective operations, half-isotonic saline for those with acute abdominal conditions, who have lost electrolytes in the gastro-intestinal secretions, and isotonic saline and/or one-sixth molar (isotonic) sodium lactate (with or without dextrose) for cases with severe losses from copious vomiting or diarrhœa, should be prescribed at this stage. Plasma and/or blood should always be given to anticipate shock during operations on severely ill patients, and during all major operations.

Daily Infusion.—

Amount.—This is based upon the usual oral intake of infants and young children, being higher for infants relative to their size. Thus 2½–3 oz. (75–90 ml.) per lb. body-weight daily are given to babies weighing less than 15 lb., but after this the amount is raised more slowly in proportion to weight (*Table II*), remaining roughly proportional to surface area and metabolic rate. The volumes suggested are larger, approximately 2·4 l. per sq. m. body surface,

than those recommended in current pædiatric text-books of surgery (Gross, 1953), but are similar to those used in medical practice (Medical Research Council, 1952; Dodd, 1954; Young and Rogers, 1954). The suggested volumes are well tolerated and must be used to ensure rehydration and a high urine output during the first twenty-four hours. Any surplus offsets the effects either of sudden losses of body fluid such as occur from 'pooling' in the bowel, or of changing the site of the infusion, which inevitably reduces the actual, compared with the prescribed, amount of fluid given.

The volumes suitable for newborn infants on the first day of life are calculated as one-seventh of the requirements of older infants of the same weight and are increased by this fraction on successive days, but these amounts must always be augmented for infants whose dehydration has not been fully relieved by the initial infusion and for those sustaining persistent abnormal losses.

Composition.—During operations solutions without potassium are given, but the usual fluid for daily infusion is one-fifth isotonic saline (sodium and chloride 30 mEq./l.) in 5 per cent dextrose, to which

Table II.—The Variations in Amount of Solutions for Infusion in 'Initial' and Subsequent 24-hr. Periods

Age in Years	Weight in Lb.	Initial Infusion (4–6 hr.) Volume ml.	Daily Infusion (24 hr.)
Less than 2	4	80–120	360
	5	100–150	450
	6	120–180	450–510
	7	140–210	525–595
	8	160–240	600–680
	9	180–270	675–765
	10	200–300	750–850
	11	220–330	825–935
	12	240–360	900–1020
	13	260–390	975–1105
	14	280–420	1050–1190
	15	300–450	1125–1275
	15–28	300–850	1250–1750
2–5	28–41	500–1000	1500–2000
5–7	41–54	750–1250	1750–2250
7–10	54–72	900–1500	2000–3000
More than 10	More than 72	More than 1000	More than 3000

potassium chloride or acetate is added to give a concentration of about 20 mEq./l. of potassium. Potassium chloride is used for the more frequent cases with losses from the upper bowel, and the acetate for the few losses from the lower bowel and those with an acidosis. The acetate is also used where there is œdema due to over-retention of sodium chloride, so that the sodium displaced from the cells by potassium may be excreted instead of forming sodium chloride which would increase the œdema (Gamble and others, 1951).

Protein, or protein digests with 5 per cent dextrose, either as plasma diluted to quarter strength or as 1¼ per cent amigen (Footnote, *Table I*), are given to infants in volumes to supply about 1·0 g. protein per lb. body-weight daily, but they are seldom needed for older children. By adding potassium salts the electrolyte composition of these solutions can be raised to that of the simple dextrose electrolyte solutions.

The amounts of fluid and electrolytes (sodium, potassium, and chloride) received by patients

Table III.—The Daily (24-hr.) Amounts of Fluid and Electrolytes (total and per kg. body-weight) from the Infusions prescribed according to the Scheme

Age in Years	Mean Weight lb. and kg.*	Initial Infusion	Daily Intake Total and per kg.† body-weight			
			Vol. ml.	Na mEq.	K mEq.	Cl mEq.
Less than 2	Less than 15 (7)*	As required by individual case	(175)	(5·25)	(3·75)	(9)
	21 (9·5)		1500 (160)	45 (4·8)	33 (3·5)	78 (8·3)
2–5	34 (15)		1750 (116)	53·5 (3·5)	38·5 (2·6)	92 (6·1)
5–7	47·5 (21·5)		2000 (93)	60 (2·8)	44 (2·0)	104 (4·8)
7–10	63 (28·5)		2500 (88)	75 (2·6)	55 (1·9)	130 (4·5)
over 10	over 72 (33)		3000 (<90)	90 (<2·7)	66 (<2)	156 (<4·7)

* Figures in parentheses in column 2 indicate weight in kilogrammes.

† Figures in parentheses in columns 4–7 indicate amount per kg. body-weight.

according to weight and age from the solutions described (*Table* I), if administered at the standard rates (*Table II*), have been summarized in *Table III*. It shows that the infants who tend to lose more than the older children receive relatively larger amounts of electrolytes from the higher volumes of fluid prescribed. *Table III* is used as the standard schedule for comparing these amounts with those given to an individual patient (*see also Table IV*).

Replacement Infusions.—Amounts to be given for replacement of losses from the gastro-intestinal tract are assessed from: (1) the volume of aspirated gastro-intestinal fluid; (2) the degree of abdominal distension; and (3) recurring dehydration and oliguria. Since the amounts of electrolytes derived from the daily infusions (*Table III*), except during the first week of life, exceed those required for maintenance of fluid equilibrium in normal circumstances, it may not always be necessary to replace small losses from the bowel, but larger losses, and even smaller losses in the newborn, should be replaced by isotonic saline at regular intervals of not longer than six hours. The onset of gross abdominal distension due to 'pooling', or of clinical dehydration and oliguria, indicates that a volume exceeding that of the measured aspirations should be given immediately. Potassium salts in concentrations similar to those for the daily infusions are added to replacement infusions if these are keeping pace with the losses, but if there is

oliguria, they must be withheld until the urine output improves. Hypotonic rather than isotonic saline is used to replace the increased losses from the skin and lungs in patients with pyrexia and pulmonary disease.

PRESENT INVESTIGATION

The treatment was prescribed by the house officer advised by a senior resident medical officer who was already experienced in the application of the scheme. Special circumstances were discussed with a consultant member of the staff, and the later treatment of these patients managed by a team including the authors. The application of the scheme to the needs of patients with a complicated clinical course has not been completely uniform, since several doctors took part in prescribing the treatment and in making the observations, and experience gained in early cases was incorporated into the treatment of the later ones in the series.

Antibiotics, usually penicillin and streptomycin intramuscularly and/or terramycin intravenously, were given in high dosage, and if intravenous infusions were continued for longer than forty-eight hours, vitamins B, C, and K were given intramuscularly on alternate days. The infusions were begun before operation if the patient was clinically dehydrated or about to have a severe operation, immediately after operation if it had been unexpectedly extensive or there was gross disease of the bowel and peritoneal cavity, or at a later stage if the patient did not tolerate oral feeding.

Clinical Observations.—The patients who had been dehydrated were closely observed throughout the initial phase of their treatment. Thereafter they were all examined three or four times each day in order to detect the onset of such serious complications as paralytic ileus. Clinical appraisal to assess probable changes in the water and electrolyte equilibrium of the body included examination for (1) variations in general well-being; (2) persisting or recurring dehydration and oliguria; (3) cerebral signs suggestive of water intoxication; (4) œdema or 'masked œdema' with gain in weight (many of the infants were weighed at frequent intervals throughout the period of infusion); (5) cardiovascular dysfunction, such as filling of the neck veins, and liver enlargement attributable to overloading of the circulation, or peripheral circulatory failure in shocked patients; (6) muscular weakness and unexpected abdominal distension suggesting potassium depletion; or (7) irregularities of the heart action suggesting potassium intoxication. The last two were to be confirmed by the serum potassium level and by an electrocardiogram.

Table IV.—CHART USED FOR SUMMARY OF FLUID THERAPY FOR AN INDIVIDUAL PATIENT

Name *Diagnosis* *Age* *Weight*

Ward *Operation* *Estimated daily requirement* Vol. ml.
Sodium mEq.
Elective or Emergency Potassium mEq.

DATE AND TIME	ORAL		ASPIRATIONS Vol. ml.	I. V.		TOTAL			
	SOLUTION	Vol. ml.		SOLUTION	Vol. ml.	Vol. ml.	Total mEq.	Per Kg. mEq.	Protein g.
INITIAL INFUSION							Na K Cl	Na K Cl	
DAY 1							Na K Cl	Na K Cl	
DAY 2							Na K Cl	Na K Cl	
DAY 3							Na K Cl	Na K Cl	

Intake.—The intake of fluid and electrolytes was reviewed at the end of each 24-hour period or more often in complicated cases, the amounts being compared with those prescribed (*Table III* or variants), and recorded on a chart (*Table IV*). Thus a summary of the preceding fluid therapy was always available and if a patient's condition deteriorated, contributory defects in his previous treatment could be appreciated before prescribing further treatment.

Output.—The losses from the gastro-intestinal tract were collected by aspiration, measured, and the amounts recorded. The output of urine could seldom be measured, and it had to be assessed by the appearance of occasional specimens, the frequency of micturition, and the extent of bed-wetting. A scanty amount of darkly coloured urine leaving a deposit on standing indicates oliguria. During the latter part of this investigation the specific gravity of each specimen of urine passed during the 24-hour period following operation was measured whenever possible, in order to observe the incidence of an antidiuretic phase following operation.

Plasma Electrolyte Levels.—The levels of sodium, potassium, and chloride and/or carbon dioxide combining power (hereafter referred to as bicarbonate) in the plasma were estimated daily, the first blood-sample being taken about twelve hours after the beginning of the first daily infusion. In

very ill patients a sample was also examined prior to or during the initial infusion. The concentration of electrolytes in the plasma can seldom be used as a guide to the requirements for replacement, since high levels are frequently found in dehydrated patients with a history of vomiting and/or diarrhœa, despite an absolute deficit of electrolytes (Cooke, 1955), and low levels may be due to an increase in the volume of the body fluids with or without changes in the amount of electrolytes they contain. In the absence of fluid retention, however, low levels indicate deficiencies with risk from clinical sequelæ. The levels were estimated in the present study principally to show whether the prescribed amounts of potassium carried any risk from potassium intoxication, and whether those of sodium, potassium, and chloride were adequate to prevent severe depletion, particularly for patients receiving prolonged infusions.

Measurement of Intake and Output.—The complete daily intake of water and electrolytes and the output in the urine and from the gastro-intestinal tract were measured in a few of the children expected to need prolonged periods of intravenous fluid therapy. The difference represents the daily amounts retained together with the losses from the skin and lungs. The cumulative amounts of electrolytes can be regarded as approximately equal to the deficits incurred before operation. The collections for 12-hour periods were measured and stored for analysis. The infusion fluids were standard solutions prepared by the hospital pharmacist and the intakes were calculated.

Laboratory Technique.—Electrolyte levels were usually determined on heparinized plasma, sometimes on serum. Sodium and potassium were estimated by the flame photometer using 0·05 ml. for each determination, chloride by Schales and Schales' method (1941) on 0·01 ml. using an Alga micrometer syringe, and the carbon dioxide capacity on 0·1 ml. by the manometric method of Van Slyke and Neill (1924). The concentration of sodium and potassium in urine and aspiration fluid were also determined by flame photometer, chloride by titration with silver nitrate solution, and nitrogen by the micro-Kjeldahl method (Levin, Oberholzer, and Whitehead, 1950). The urinary specific gravity was determined by weight.

CLINICAL MATERIAL

Observations were made on a consecutive series of 97 children aged 1 month to 13 years and 23 newborn infants, each of whom required intravenous therapy, during the 3-year period from April 1954 to 1957.

Children aged 1 Month to 13 Years.—Seventy-six of the children were suffering from acute abdominal conditions and 21 underwent major elective surgery.

Acute Abdominal Conditions.—These cases were arranged according to age and the clinical categories of inflammatory conditions (39), pyloric stenosis (4), intussusception (8), and acute intestinal obstruction (25).

Inflammatory conditions: Thirty-five children had acute appendicitis with peritonitis, 2 had infected mesenteric cysts, and 2, aged less than three months, generalized peritonitis following abdominal surgery. During the period of investigation a greater number of older than younger children had operations for acute appendicitis, but more of the younger ones required intravenous therapy owing to a higher incidence of generalized peritonitis. Thus, among the 196 patients requiring operation, 113 were over 7 years of age, 34 between 5 and 7 years, and 49 between 2 and 5 years of age, and intravenous therapy was given to 11 (10 per cent), 9 (25 per cent), and 15 (30 per cent) respectively. This is in accord with Mason Brown's (1956) observations concerning the age incidence of complications from appendicitis in Edinburgh.

Five of the 35 children suffering from appendicitis and both infants with generalized peritonitis were clinically dehydrated and therefore given their intravenous infusions before operation. The mesenteric cysts were found at laparotomy and both required extensive surgery for their removal. The intravenous infusion was started during the operation in one, and just after in the other case.

Pyloric stenosis and intussusception: All 4 infants with congenital hypertrophic pyloric stenosis were under 3 months of age, and 7 of the 8 with intussusception under 2 years. Intravenous therapy for severe dehydration was given before operation to 3 with pyloric stenosis and to 1 for post-operative vomiting. The infusion was begun pre-operatively in 6 cases of intussusception in anticipation of surgical shock, and post-operatively in 1 for severe vomiting, and in another following a difficult reduction. During the period of the investigation 56 other babies with pyloric stenosis and 15 with intussusception were treated surgically without intravenous therapy.

Acute obstruction: Twenty of the 25 episodes occurred in patients with congenital abnormalities of the bowel and many had acute obstruction superimposed upon chronic obstruction of the bowel. Eight had Hirschsprung's disease, and the 12 others had late complications of imperforate anus (4), rectovaginal fistula (2), volvulus (2), meconium ileus (2), strangulated inguinal hernia (1), and ileal atresia (1). The 5 cases of acute obstruction from acquired disease were due in 3 to adhesions, in 1 to Henoch's purpura, and in another to a grossly dilated loop of ileum with thickened mesentery. Acute obstruction usually occurred before the age of 2 years in patients with congenital abnormalities of the bowel, whereas in all those with acquired disease it occurred later. Manifest dehydration was present in 17 of the 25 cases, and their infusions were begun before operation; one other became dehydrated later and should have had an infusion earlier.

Major Elective Surgery.—Fifteen of the 21 cases had operations on the bowel for congenital abnormalities (including Swenson's operation and the reconstruction operation for malformation of the anus and rectum), and 6 on the renal tract (plastic procedures (3) and nephrectomy for renal tumour (3)). An infusion was always started before the operation.

Newborn Infants.—Twenty-three babies, 8 of whom weighed less than 5 lb. at birth, had congenital intestinal obstruction. Their age at presentation ranged from 1 to 10 days, and many had associated congenital abnormalities. The causes of obstruction were: (1) *Duodenal atresia* in 7 babies (including 5

mongols, 2 with congenital malformations of the heart); all but 2, both less than 2 days of age, were dehydrated and further to the operation of duodeno-jejunostomy, 1 required the removal of inspissated meconium, and another a colostomy for imperforate anus. (2) *Ileal atresia* in 1 patient who was already clinically dehydrated on the second day and very extensive resection of the small bowel was necessary prior to an anastomosis. (3) *Meconium ileus* in 4 infants, 2 aged 2 days, one aged 4 days, and one 5 days old, both of the latter being dehydrated; an additional case has been described under duodenal atresia. (4) *Exomphalos* in 4 babies, each aged 2 or 3 days, 3 of whom had other gross abnormalities, namely, transposition of the great vessels, ectopia vesica with ileal atresia, and herniation of the whole of the liver out of the abdominal cavity, which on being replaced caused obstruction of the duodenum and inferior vena cava. None of these infants was dehydrated on admission and the intravenous infusion was started after the operation in two of them. (5) *Hirschsprung's disease* in 2 babies, 1 aged 3 days and the other 10 days; both were dehydrated and the younger child also had bladder-neck obstruction, megalo-ureters, and hydronephrosis and multiple perforations in the small bowel. (6) *Imperforate anus* in 3 cases, all younger than 3 days and not dehydrated; one was a mongol and a fourth case has been described under duodenal atresia. (7) *Other causes* of partial obstruction in 2 infants, 1 aged 5 days, from the ligating of a Meckel's diverticulum within the umbilical cord, and the other, aged 7 days, from an inguinal hernia; the latter was dehydrated.

RESULTS

Clinical Progress.—

Children aged 1 Month to 13 Years.—Ninety-three of the 97 children survived the post-operative phase and only 1 died later. Progress was uneventful in 54, but complicated by episodes of clinical deterioration in 39, occurring either early (during the initial infusion) or later (during subsequent daily infusions) in the course of treatment. Since these might have been due to the disease *per se*, the part played by electrolyte imbalance was assessed individually.

Deaths: Each of the 5 children who died had intestinal obstruction, in 1 due to an intussusception. Four were gravely ill and shocked before operation. Two had gangrenous bowel which could not be removed, but the two others might have been saved by more intensive resuscitative therapy with blood and plasma before and during operation. One died a week after a colostomy for Hirschsprung's disease and autopsy showed widespread necrosis of the small bowel.

Complications: Four children had *convulsions*. They occurred early in 2 who had been severely dehydrated; later in 1 after the loss of large amounts of gastro-intestinal contents from paralytic ileus after an elective operation, and in another, a case of undiagnosed cystic fibrosis of the pancreas with acute obstruction due to inspissated fæces, whose losses had probably been enhanced by high concentration of salt in the sweat (Di Sant'Agnese, Darling, Perera, and Shea, 1953). Water intoxication following sodium depletion and failure to replace the salt losses were the causes of the convulsions in these four cases. All recovered from them, but one, a mongol with congenital heart disease, developed gross peripheral œdema and later was judged to have reduced mental ability. Another who had circulatory collapse from the outset of treatment also developed peripheral œdema. The records of two of the children who had convulsions are now described in greater detail.

Case 1.—Geoffrey S., aged 5 years 5 months, weighing approximately 22 kg., had had abdominal pain for 4 days, with vomiting at the onset continuing for 2 days, and diarrhœa with frequent and copious stools throughout. The child had become severely dehydrated and his abdomen grossly distended. He had signs of acute appendicitis, and at operation an inflamed appendix was removed. An intravenous infusion of 5 per cent dextrose in half isotonic saline had been started before operation, and after 1½ hours, when 500 ml. of this solution had been given, the patient began to have generalized convulsions. The plasma concentrations of sodium and potassium before the infusion began were later found to be 127 and 3·3 mEq./l. respectively. With suitable treatment, the subsequent progress of this patient was uneventful. The history of large losses from the bowel is the clue to his deficit of electrolytes, and if this had been fully appreciated he would have been given an initial infusion containing isotonic saline.

Case 2.—James F., 5 weeks old, weighing 3·5 kg., had an increasingly distended abdomen for 24 hours, and had vomited bile-stained fluid on several occasions; 184 ml. of gastro-intestinal fluid had been aspirated, but only 100 ml. of 5 per cent dextrose in half isotonic saline had been given by subcutaneous infusion. The baby had become very dehydrated, his temperature was 103° F., his abdomen grossly distended, and he had signs of peripheral circulatory collapse. An intravenous infusion was started immediately, and 3 hours later a colostomy made for relief of acute intestinal obstruction due to Hirschsprung's disease.

At the beginning of the infusion the plasma sodium was normal, 142 mEq./l. After 370 ml. of 5 per cent dextrose in half isotonic saline and 85 ml. in one-fifth isotonic saline had been given in 9 hours the baby had generalized convulsions. During the infusion no urine was passed and 150 ml. of gastro-intestinal fluid was aspirated. After isotonic saline had been given by infusion the urinary output improved, but 12 hours later sacral œdema developed. The plasma sodium was then only 124 mEq./l., but there were no further convulsions. Subsequently the clinical condition of the baby improved, but the obstruction persisted, and one week later he died from massive necrosis of the bowel.

A sudden onset of acute obstruction with high gastro-intestinal losses caused the severe dehydration with salt depletion, followed by circulatory collapse and impaired renal function in this child. The initial infusion of half isotonic saline must have diluted and expanded the volume of his extracellular fluids to a dangerous extent. Similar circumstances are now regarded as an indication for an initial infusion containing isotonic saline or plasma.

Ileus and vomiting from peritonitis complicated the course of 38 cases, but in 16 the ileus was transient, being relieved within 48 hours. The management of one of these, *Case* 3, Martin A., aged 3 years 1 month, is summarized in *Table V*. He was suffering from acute appendicitis with generalized peritonitis and had had fever for 1 day, abdominal pain for 2, and vomiting for 3 days. Intravenous infusion was begun immediately after operation. It should be noted that isotonic saline was used for part of the initial infusion and for replacement on day 1. Despite this, the plasma

sodium levels were low, 130 and 132 mEq./l., on days 1 and 2, but the other electrolyte levels were normal.

Twenty-two patients (20 with ileus and 2 with vomiting from peritonitis) needed a more prolonged period (exceeding 48 hours) of intravenous therapy. Persistent ileus occurred in only 3 of the 39 children with inflammatory lesions, whereas it occurred in 10 of 22 with acute obstruction of the bowel, and in 4 of 15 during recovery from elective operations for congenital abnormalities of the bowel. These malformations may have rendered them more susceptible to ileus, for Nixon (1956) has shown that after chronic partial obstruction is relieved, the bowel functions poorly for some days. Only one patient developed ileus after an operation on the renal tract.

Table V.—MANAGEMENT OF A CASE* WITH ILEUS AND VOMITING FROM PERITONITIS (*Case* 3)

	INITIAL INFUSION FOR 5 HR. AFTER OPERATION	DAY 1	DAY 2
Clinical	Febrile, dehydrated	Well hydrated. Good urine flow	
		Ileus	Ileus relieved
Aspirations ml.	31	91	46
Solutions by infusion (ml.)	N.S.† 350 N/2‡ 680	N/5§ + KCl‖ 1670 N.S. 120	N/5 + KCl 800 *Oral* 684
Total vol. ml.	1030	1790	1484
mEq. Na	120	68	24
mEq. K	0	35	16 (+ oral)

* Martin A., aged 3 years 1 month; estimated daily requirements: Vol. 1750 ml. Na 53 and K 38 mEq.

† N.S., Isotonic sodium chloride solution.

‡ N/2, half isotonic sodium chloride solution (5 per cent dextrose).

§ N/5, one-fifth isotonic sodium chloride solution (5 per cent dextrose).

‖ + KCl, 20 mEq. potassium chloride added per litre of solution.

Œdema was not observed except in the two cases already described. There were occasional instances of recurring dehydration with oliguria, due either to 'pooling' of gastro-intestinal contents without adequate replacement, or an accidental fall in the rate of fluid administration. Clinical signs suggesting either potassium depletion or intoxication were not observed.

Newborn Infants.—Twenty-two of the 23 infants with congenital intestinal obstruction survived the early post-operative phase, but 4 died subsequently.

Deaths: The patient with Hirschsprung's disease with multiple perforations of the small bowel and other abnormalities died immediately after operation. Four other infants died who had received intravenous therapy judged to have been satisfactory for some days, but they had such severe disease that they could not have been expected to survive. Death was due to (1) meconium ileus causing unrelieved obstruction in a case of duodenal atresia, on the sixth day; (2) transposition of the great vessels in a case of exomphalos, on the twelfth day; (3) hæmorrhagic pneumonia complicating the exomphalos with gross herniation, on the fourteenth day; and (4) meconium ileus which remained obstructed until a high ileostomy was performed on the twenty-eighth day, the child dying eight days later.

Complications: Progress was straightforward, with early recovery of bowel function in only 5 of the 22 infants. Intravenous therapy was given to 17 for 4 days or longer and to 9 for over a week. Delay in recovery of bowel function was to be expected in those with hypotrophic bowel, and, in the 13 of these cases which survived, in 6 the delay lasted more than a week after operation, in 1 the interval being 3 weeks. Most of these infants appeared vigorous and were sometimes hungry despite unrelieved obstruction and 'pooling' of gastro-intestinal secretions in the upper bowel. Only 2 infants developed manifest œdema, due in one to obstruction of the inferior vena cava by the liver and in the other—an infant with congenital heart disease—to circulatory difficulties following a period of gross sodium depletion. None had convulsions. Loss of large volumes of gastro-intestinal fluid by aspiration and from 'pooling' sometimes led to temporary phases of oliguria. None had clinical manifestations of either potassium deficiency or intoxication.

Renal Function.—

Urine Flow in Response to Initial Infusion.—The time from the start of the initial infusion until a free flow of urine was passed was recorded in 89 children of the older age-group and in the 22 surviving newborn infants. The results have been analysed according to each patient's state of hydration and the rate at which his infusion was given, the response being regarded as satisfactory if urine was passed within six hours.

Older infants and children: Thirty with slight, and 15 with manifest, clinical dehydration received infusions at a rate within the range of the schedule, and all but 2 of the latter passed urine within six hours; also each of 6 severely dehydrated patients given infusions at a faster rate passed urine within this time. In contrast, only 9 of 15 slightly, and neither of the 2 more severely, dehydrated children given infusions at a slower rate passed urine so soon. Twenty-one children received infusions, usually of blood or plasma, at a slow rate during the course of major elective operations; 17 of these did not pass urine during the six hours thereafter, but as they had not been dehydrated, this delay did not matter.

Newborn infants: Five of 12 slightly dehydrated babies received infusions at the scheduled rate and all passed urine within six hours; 7 received them at a slower rate and 5 passed urine within this time. Six of 10 severely dehydrated infants received infusions at the scheduled rate, and 4 at an even faster rate, but only 2 of each group passed urine within six hours. This delay before passing urine reflects the severity of dehydration frequently encountered in young infants with intestinal obstruction.

Dilution of Urine following Operations.—Urine with a specific gravity of less than 1010 was obtained within 24 hours from 18 of 22 consecutive patients in the older age-group in whom it was measured. It lay between 1015 and 1020 in 3 patients who had been 'pooling' intestinal contents, and in 1 who had become profoundly collapsed during and after the removal of a large infiltrating adenocarcinoma of the kidney. Although the specific gravity of the urine was not always measured, the majority of the other cases in the series, including the newborn infants, are known to have passed dilute urine in high volume at this stage.

The results indicate that no antidiuretic response to operation occurred among this series of cases. It therefore seems safe to assume that the fluid intake of infants and children who have oliguria should be increased during the post-operative phase.

Urine Flow during Prolonged Periods of Intravenous Therapy after Operation.—Fig. 276 illustrates the variations in the output of urine in relation to intake and losses by aspiration in one of the cases of the series.

Case 4.—James B., aged 10 months, weight 8 kg., had had mild diarrhœa, increasing abdominal distension, colicky abdominal pain, and fever up to 103° F. for 4 days. He had been drinking well without vomiting and

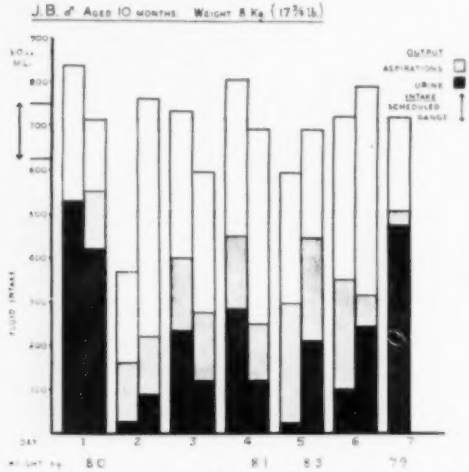

FIG. 276.—*Case* 4. The intake and the output of urine and of gastro-intestinal aspirations during successive 12-hour periods in a patient with ileus lasting nearly a week.

was not dehydrated, but his abdomen had become tensely distended and tender. Laparotomy revealed a very large mesenteric cyst which was dissected from the bowel. During the operation an intravenous infusion of half-strength plasma to anticipate shock was begun. Although paralytic ileus developed 12 hours later and persisted for 6 days the child's general condition remained good throughout. It will be seen that the volume of urine was high during the first 24-hour period. Towards the end of this time the abdomen again became distended and the volume of aspirations increased, and during the first half of the second day the intake was inadvertently diminished. Consequently, the output of the urine was very low and the patient became somewhat dehydrated.

The urine flow improved when a rapid infusion of half isotonic saline was given, and in spite of high losses from the gastro-intestinal tract was then fairly well maintained until the first half of the fifth day. The daily intake of fluid was then adequate according to the schedule, but the abdomen suddenly became more distended and a 'pool' formed which later became apparent from the volumes of fluid being aspirated. During the second half of the fifth day the aspirations were still large, but the degree of abdominal distension was unchanged and the urine output improved. On the sixth and seventh days it increased, reflecting a return of bowel function with reabsorption of the 'pool' and release of fluid for excretion. During most of the period of fluid therapy the urine output was being well maintained, so that the volumes aspirated from the bowel were replaced with safety by isotonic saline solution containing potassium salts.

The uniformity of the body-weight throughout shows that fluid was not retained. The plasma electrolyte levels were consistently normal, except for a somewhat low potassium level, 3·8 mEq./l., on the fourth day (*see below*).

Plasma Electrolyte Levels.—

Children aged 1 *Month to* 13 *Years.—*

Plasma sodium levels: A level below 130 mEq./l. is regarded as an indication of danger from water intoxication.

During the first 24*-hour period*, the plasma sodium was estimated in 68 children and was below this level in 20. The estimated losses relative to replacement have been compared in children with low and those with normal levels. Seventeen of the 20 had acute abdominal conditions with large gastro-intestinal losses, either before or after operation, 3 of whom had developed rapidly increasing abdominal distension after elective operations, but only 2 had been given isotonic saline at any stage. In contrast, among the 48 children with normal levels, 16 of 30 with similar high losses had received isotonic saline solution for replacement, while 18 others had not had significant losses. All the children with high losses should have received more salt as isotonic saline early in their treatment. This was not given, owing in some cases to errors of judgement in the application of the scheme, and in others because early in the investigation the significance of 'pooling' of gastro-intestinal secretions was not fully appreciated.

At later stages of treatment: Six of the 22 children receiving prolonged intravenous infusions had low plasma sodium levels occurring on the second or third day. Three were known to have had low levels previously, and all had increasing abdominal distension and losses of gastro-intestinal fluid by aspiration. The administration of isotonic saline for replacement had been delayed in 5 cases; in the other it had been given in volumes approximately equal to the amounts aspirated, but no allowances for a large gastro-intestinal pool was made.

Plasma potassium levels.—A concentration below 3·8 mEq./l. is regarded as an indication of a deficiency.

During the first 24*-hour period* the plasma potassium was estimated in 78 children and none higher than 7 and only 4 between 6 and 7 mEq./l. were found. The levels were normal in 16 children after elective surgery. Four with hypochloræmic alkalosis (bicarbonate over 40 mEq./l.) due to congenital pyloric stenosis had the expected low levels, and also 12 among 58 with acute abdominal conditions, but these patients had normal or near normal concentrations of chloride and of bicarbonate in the plasma. The incidence of low levels was the same in patients with inflammatory and obstructive conditions, but was greater in those who had been dehydrated. Thus 7 (30 per cent) of 23 with manifest dehydration and 5 (15 per cent) of 33 children with slight dehydration had low levels after this was relieved. Three of the 12 children with low levels and 11 of 43 with normal levels developed ileus. The incidence was therefore no greater among the children with low than among those with normal potassium levels at this stage. After receiving daily infusions containing potassium on the first and subsequent days, 9 of those with earlier low levels were re-examined

and found to have normal levels, 8 by the second and 3 by the third day.

At later stages of treatment, the concentration of potassium in the plasma was estimated daily in 19 of the 22 children who received prolonged intravenous infusions. It was high (8 mEq./l.) in 1 on one occasion (*see below*), and low in 3 during the third and 7 during the fourth 24-hour period; thus more than half the children with persistent intestinal obstruction were at risk from potassium depletion, which might have progressed to give clinical manifestations if they had not been receiving potassium salts systematically. Some had received less potassium than planned, owing to periods of oliguria and consequent withdrawal, but most had been given the scheduled amounts throughout their course.

aspiration were increasing, the daily infusion was not augmented at once by replacement infusions. Consequently the patient became dehydrated and his sodium depletion was enhanced. The plasma sodium level was only 122 mEq./l. on day 2, representing a very severe degree of depletion in a dehydrated patient. The level then remained low in spite of replacement of aspirations by infusions of 400, 700, and 300 ml. of isotonic saline, during the second, third, and fourth daily periods. This indicates the magnitude of the losses by 'pooling' for which no allowance was made in assessing the volume to be replaced. Since the urine output was scanty on the second and third days, and the plasma potassium high on the second day, very little potassium was given by infusion, and by the fourth day the plasma potassium level had dropped to 3·5 mEq./l. It will be seen that in spite of large replacement infusions the total volume of fluid given daily was no higher than that of the scheduled

Table VI.—Management of a Case* with Persistent Intestinal Obstruction (*Case* 5)

	Initial Infusion 6 hr. Pre and Post Operation	Day 1	Day 2	Day 3	Day 4	Day 5
Clinical	Gross abdominal distension Severe dehydration	Abdominal distension increasing Dehydration relieved	Very gross abdominal distension Dehydrated Ileus	Dehydration relieved	Abdominal distension less	Ileus relieved Partial oral feeding
Aspirations (ml.)	0	190	370	320	130	0
Intake (mEq.)	Vol. 1100 Na 84 K 0	Vol. 2201 Na 68 K 47	Vol. 1835 Na 129 K 27	Vol. 2155 Na 172 K 6	Vol. 2175 Na 114 K 47	Vol. 950 Na 46 K 29
Urine output		Good Adequate	Poor Scanty	Poor Scanty	Good Adequate	Good
Plasma (mEq. l.)		Na 127 K 5·9 Cl 90	Na 122 K 8 Cl 90	Na 117 K 5·6 HCO_3 29·5	Na 124 K 3·5	Na 127 K 4·1

*Colin T., aged 5 years 10 months, weight approx. 20 kg.; estimated standard daily requirements: vol. 2000 ml. Na 60 mEq.; K 44 mEq.

Plasma chloride and bicarbonate levels: These were not estimated as regularly as those of sodium and potassium, but one or the other was usually estimated daily in the complicated cases. Generally a low chloride accompanied a low sodium level, and only 1 other case had gross hypochloræmic alkalosis comparable with that of 2 of the patients with pyloric stenosis (bicarbonate levels 36 and 39 mEq./l.). The bicarbonate levels were between 30 and 35 mEq./l. in 12 additional patients at some stage, but were usually normal on the following day.

Unusually severe deviations from normal electrolyte levels were found in the patient described below and in *Table VI* who had persistent intestinal obstruction and a hazardous post-operative course.

Case 5.—Colin T., aged 5 years 10 months, weighing approximately 20 kg. (*Table VI*). Swenson's operation had been performed 3 years previously. Bowel function had been satisfactory until 3 days before admission when the boy became constipated and vomited frequently. He became very dehydrated and had gross abdominal distension. The terminal colon was found to be obstructed by 'kinking', and this was relieved. Subsequent progress was complicated by a long period of ileus, with ultimate recovery.

The history and clinical findings at the outset suggested a considerable loss of electrolytes. Hydration was improved by the initial infusion of 5 per cent dextrose in half isotonic saline, but the plasma sodium level was low, 127 mEq./l. on day 1. Although abdominal distension and the volumes of gastro-intestinal fluid obtained by daily intake. The low fluid intake was due to technical difficulties, but it may have reduced the risk of water intoxication when the serum sodium levels were low. The later management of this case would have been easier if the sodium deficit had been more completely repaired at the outset.

Newborn Infants.—

Plasma sodium levels: These were low, below 130 mEq./l. in 8 of the 22 infants, 6 of whom were less than a week old. Five had low levels a few hours after their infusion was started, and 3 at a later stage.

Some, like the older children with intestinal obstruction, had therefore become depleted from persistent losses which had been inadequately replaced.

Plasma potassium levels: Only 1 infant had a high level, 8 mEq./l., which occurred on the day intravenous fluids were discontinued. This rise was probably due to hæmolysis of red cells with liberation of potassium as the baby became jaundiced.

Ten infants had low levels, between 2·8 and 3·8 mEq./l., 3 during the first twenty-four hours and 7 at a later stage. Thus some were at risk from depletion although they had received potassium in the small amounts recommended for this age.

The response to treatment of 2 patients with duodenal atresia who were operated on within a few hours of admission illustrates the serial findings for this age-group.

Case 6.—George H., aged 3 days, weight 4 lb. 10 oz. (*Fig.* 277), had been vomiting and was severely dehydrated. An intravenous infusion of 5 per cent dextrose in half-isotonic saline was begun before, and continued with plasma during, the operation. Nearly 70 ml. per lb. (150 ml. per kg.) body-weight was given before urine was passed. Thereafter the intake of both fluid and sodium was larger than the scheduled amounts, the sodium sometimes being twice as great owing to the large replacement infusions of isotonic saline necessary. However, the baby did not become œdematous or gain weight excessively, and his plasma sodium levels remained normal. The scheduled amount of potassium was given after the first day. From the third day onwards, plasma and/or casein hydrolysate solutions were given. The plasma potassium and chloride levels were normal throughout.

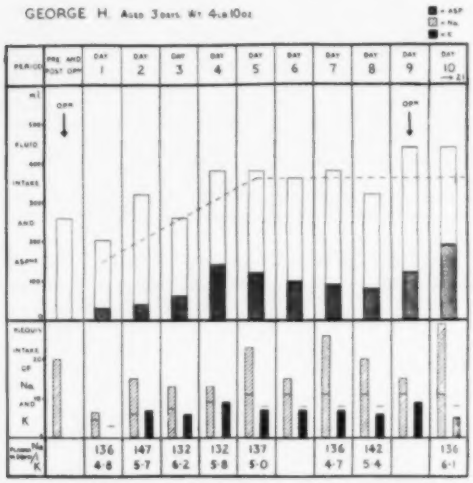

FIG. 277.—*Case* 6. The daily intake of fluid and electrolytes is compared with the schedule, and the volume of gastro-intestinal fluid aspirated is superimposed. The variations in the plasma sodium and potassium levels are also shown. Scheduled volume of fluid ml. — — —. Scheduled sodium and potassium in mEq. —.

A second operation was performed, but intestinal obstruction persisted, with continuous aspirations of gastro-intestinal contents until the nineteenth day, intravenous therapy being given until the twenty-first day. The child's general condition was satisfactory, and subsequently he gained well on oral feeding, weighing 7 lb. 8 oz. six weeks later.

Case 7.—Susan L., aged 2 days, weight 4 lb. 4 oz. (*Fig.* 278), had been vomiting, but was not dehydrated. An intravenous infusion with 5 per cent dextrose in half isotonic saline was begun before, and continued with blood and plasma during, the operation. As the volumes for the initial and the earlier scheduled daily infusions were at first very small, they were given by intermittent injections into a venous cannula left in situ. Potassium was omitted until a continuous infusion was started on day 4.

Intestinal obstruction persisted, with gastro-intestinal contents being aspirated from day 2 until day 7; oral feeding was begun on day 9. Replacement of losses by aspiration was omitted on day 2, and was only equal to the measured losses on day 3, and the plasma sodium level fell to 130 mEq./l. on day 4. Aspirations were completely replaced from day 3 onwards, the sodium intake on days 3, 4, 5, and 6 exceeding the schedule, and the plasma sodium level rose to a normal level. Since potassium was withheld until day 3, and later omitted from the solutions used for replacement infusions, the intake of potassium was below the scheduled amount throughout the first week and the plasma level fell steadily

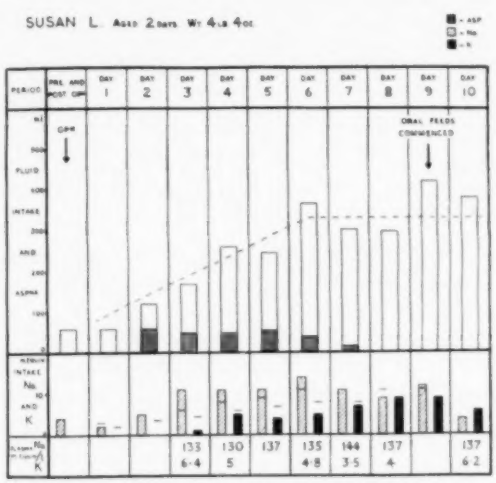

FIG. 278.—*Case* 7. The daily intake of fluid and electrolytes is compared with the schedule and the volume of gastro-intestinal fluid aspirated is superimposed. The variations in the plasma sodium and potassium levels are also shown. Scheduled volume of fluid ml. — — —. Scheduled sodium and potassium in mEq. —.

Table VII.—THE 12-HOURLY VOLUMES OF FLUID AND ELECTROLYTES GIVEN AND GASTRO-INTESTINAL FLUID ASPIRATED DURING A PERIOD OF ILEUS*

TIME	ASPIRATIONS (ml.)	INTAKE Solutions Volumes (ml.)	TOTAL INTAKE Vol. (ml.)	mEq.
Schedule		N/5† + K Cl‡ 2500	2500	Na 75 K 55 Cl 130
Initial infusion		N/2§ 1460	1460	Na 112 K Cl 112
Day 1	6	N/5 + K Cl 987	2140	Na 90 K 46 Cl 135
	164	N/5 + K Cl 943 N.S.‖ + K Cl 210		
Day 2	106	N/5 + K Cl 822 N.S. 75	2457	Na 136 K 53 Cl 189
	84	N.S. + K Cl 420 N/5 + K Cl 1140		
Day 3	28	N/5 + K Cl 1150	2625	Na 79 K 58 Cl 137
	0	N/5 + K Cl 1475		
Day 4	0	N/5 + K Cl 1185 *Oral* Orange juice 200	3310	Na 78 K 82 (approx.) Cl 135
	0	N/5 + K Cl 1425 Orange juice 500		

* *Case* 8, Michael A., weight 27 kg.; estimated daily requirements: Vol. 2500 ml. Na 75 mEq. K 55 mEq.

† N/5, one-fifth isotonic saline.

‡ + K Cl, potassium chloride 22 mEq. added to each litre of solution for infusion.

§ N/2, half isotonic saline

‖ N.S., isotonic saline.

to 3·5 mEq./l. on day 7. It rose again to normal on days 8 and 10 after the intake had been increased. The plasma levels for chloride and bicarbonate were normal throughout. Although in this case the fall in plasma potassium was transient, a stage of depletion with serious clinical sequelæ might be reached in some babies with prolonged intestinal obstruction, particularly if they had been severely dehydrated.

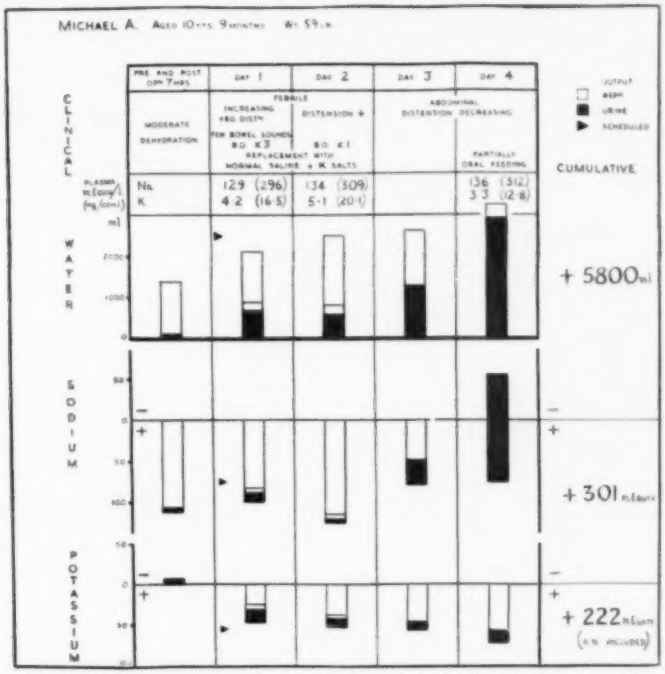

FIG. 279.—*Case* 8. The clinical course, plasma electrolyte levels, and the intake and output of water and electrolytes in a patient with prolonged ileus.

Water.—The volume of the initial and each subsequent daily infusion (and of oral fluid on day 4) is drawn above the zero line, and the output of urine and aspiration is superimposed as hatched areas. The difference between intake and output is unshaded and represents the amount retained together with the amount of unmeasured losses from the skin and lungs.

Sodium and potassium.—The intake of sodium and potassium from the initial and subsequent infusions is drawn below the zero line and the measured output in the urine and by aspiration is superimposed as hatched areas. An unshaded area below the zero line represents the amount by which the intake exceeded the measured output which includes the amount retained together with the unmeasured losses (these are very small unless a patient has been sweating profusely) and can be disregarded in *Case* 8. The retention includes the amount 'pooled' in the bowel which is temporarily lost to the body fluids. When the measured losses have exceeded the intake, a negative balance is shown by a shaded area above the line. It will be seen that this only occurred for potassium during the initial infusion in *Case* 8. An additional source of potassium from protein breakdown is available to the body and must be included in computing the overall retention of potassium. The amount is calculated by using the ratio of 3 mEq. potassium for each g. of nitrogen excreted in the urine. This amount has not been added to the daily amounts shown on the chart, but was included in calculating the cumulative balance of potassium for the 4-day period.

Chloride.—The data for the daily intake and measured output of chloride are not given, but have been used to calculate the cumulative retention recorded in the text and in *Table VIII.*

Estimates of Deficits in Individual Cases.—The fluid and electrolyte balances were more fully studied in 3 children. The case report of one patient who had the greatest estimated deficits is given below.

Case 8.—Michael A., aged 10 years 9 months, weight 27 kg., had had abdominal pain, fever, and vomiting (4–5 times) during the 48 hours before admission. He looked toxic, was somewhat dehydrated, and his temperature was 102° F. The abdomen was distended, tender, and rigid. At operation a perforated appendix with generalized peritonitis was found, and the appendix was removed.

The amount and composition of the fluids given by infusion are shown in *Table VII.* The daily requirements for this small 10-year-old boy were estimated as 2500 ml., with additions for replacement of abnormal losses. The initial infusion was given at the appropriate rate, and urine was passed after six hours. The daily infusion of 5 per cent dextrose in one-fifth isotonic saline with added potassium chloride was begun one hour later, and the total volumes given approximated to the scheduled amounts. Isotonic saline was given in volumes exceeding those of the aspirations (to allow for 'pooling') on days 1 and 2, and therefore the total intake of sodium and chloride was high on these days.

Fig. 279 shows diagrams of the measured intake and output with a summary of the clinical course and plasma electrolyte levels. The clinical condition improved when hydration had been restored and remained good thereafter in spite of ileus developing on day 1 and persisting until day 3. Oral feeding was begun on day 4.

The concentration of sodium in the plasma was low on day 1, but normal thereafter, while that of potassium was normal at first, but dropped to a low level on day 4. The plasma chloride level was normal throughout.

Water.—*Fig.* 279 shows that the volume of urine was approximately 600 ml. per day during the first two days,

which is less than expected on the scheduled intake if the extrarenal losses are normal. The rate of urine flow diminished when abdominal distension was increasing towards the end of day 1. On days 3 and 4 the bowel began to function and the 'pool' of gastro-intestinal fluid was absorbed and the volume of urine increased to an amount almost equal to the intake.

Sodium.—Up to the end of the second day the intake was 338 mEq. and since isotonic saline solution had been given for replacement, exceeded the scheduled amount on both days, but only 16 mEq. were excreted during this period. On the third day the urinary output increased and on day 4 exceeded the intake, presumably when sodium from the intestinal pool was reabsorbed.

Potassium.—Potassium was retained consistently during the 4-day period, indicating that restoration of the preceding deficit was incomplete at the end of this time.

Cumulative Retention.—During the whole period, the retention of sodium was 301 mEq., potassium 222 mEq. (including 63 mEq. derived from protein breakdown), and chloride 307 mEq., these amounts being 11, 8, and 11 mEq./kg. body-weight respectively. They are of the same order of magnitude as those recorded for infants recovering from severe gastro-enteritis and represent the losses sustained while the patient was becoming dehydrated, together with those from the skin during the period of study. If this patient, who had persisting losses from the gastro-intestinal tract superimposed upon those incurred pre-operatively, had been given less sodium and potassium, he would have been exposed to the risks of gross depletion, for even after the amounts which were given, his deficits were only slowly corrected and his plasma sodium and potassium levels were both low at some stage.

Table VIII.—THE RETENTION OF ELECTROLYTES BY THREE CHILDREN DURING RECOVERY FROM ILEUS

PATIENT	DURATION OF TREATMENT Days	CUMULATIVE RETENTION (mEq.)			DEVIATIONS FROM NORMAL VALUES IN PLASMA	
		Na	K	Cl	Na	K
Michael A.	4	301	222	307	Low day 1	Low day 4
Brian W.	4	232	163	—	Low day 1 and 2	Low day 4
Ann F.	6	70	60	100	None	None

A comparison between the deficits shown by Michael A. and those of two other children with paralytic ileus, whose ages and clinical condition happened to be similar, is given in *Table VIII*. Brian W.'s deficits as estimated by the amounts retained were of the same high order as Michael A.'s, and his plasma sodium and potassium levels also reflected a lag in replacement. Ann F.'s deficits preceding treatment appear to have been smaller, although ileus persisted for longer than in either of the other cases. Her plasma sodium and potassium levels were normal at all times.

The wide variation in the extent of the deficits cannot be deduced clinically. The two children with the greater losses would clearly have been at considerable risk from potassium deficiency if they had been treated with saline solutions without potassium salts. Michael A. might not have been expected to need potassium, since many patients suffering from appendicitis complicated by peritonitis recover without setbacks and are able to tolerate oral feeding early. The findings strongly suggest that it should be given to all such cases to cover the needs of these occasional ones who have become grossly depleted from dehydration or who develop severe and prolonged ileus of the bowel. Similar balance studies in surgical patients by Lowe, Rourke, MacLachlan, and Butler (1950) and by Holliday, Wallace, and Stapleton (1958) support this conclusion.

DISCUSSION

The plan of fluid therapy here presented, which is based upon probable losses before and during treatment, has been found suitable for children with acute abdominal conditions or undergoing major elective abdominal surgery. There need be no danger either from potassium intoxication or from over-retention of the sodium salts given in excess to ensure replacement of intestinal losses. The occasional clinical sequelæ of incorrect treatment were all due to underestimation of the requirements of sodium salts to replace previous or persistent losses. The plasma sodium and chloride levels of some of the other patients suggest that they also should have received more salt.

The scheme has been applied safely to newborn infants suffering from intestinal obstruction and is regarded as a valuable advance in the management of our cases. Recently, similar good results have been obtained in 12 more neonates, 4 with duodenal atresia (only 1 a mongol), 6 with ileal obstruction, and 1 with imperforate anus, who were given intravenous fluid therapy for periods of 2–17 days, 6 for over a week. The plan differs from that recommended by Gross (1953) and by Rickham (1957, 1958) who believe that newborn infants should be kept 'on the dry side'. Healthy breast-fed infants at first receive only small amounts of fluid and electrolytes, and they are known to be little affected by transient dehydration due to a water deficit, and to be liable to œdema if their intake of salt is increased. For this reason in the past those suffering from intestinal obstruction have often been given insufficient water and electrolytes to replace their losses before and after operation. However, since their dehydration is of the same mixed aetiology as that of older patients it should be treated according to the same principles.

Infants with persistent ileus might be expected to retain sodium from the excessive amounts given for replacement, but œdema was only observed in two cases, both with cardiovascular defects, among the present series of patients, all of whom had been given potassium as well as sodium salts at an early stage. Rickham (1957) based his assessment of the post-operative fluid and electrolyte requirements of newborn infants upon balance studies in a small series given intravenous infusions containing little or no potassium. Since, unlike adults after operation,

the potassium/nitrogen ratio in the urine was no higher than that to be expected from protein breakdown in starvation, he believes that patients of this age are unlikely to become depleted of potassium and that they require little or none by infusion. However, most of his subjects had oliguria, and some were dehydrated, and McCance and Widdowson (1954) have shown that under these conditions the potassium/nitrogen ratio in newborn infants is low. This would explain Rickham's findings and alter their implications. Furthermore, since infants with intestinal obstruction become depleted mainly from losses following dehydration and incurred during the periods of ileus, the losses due to stress at operation would be a small part of the total in this group of patients. Rickham records low serum potassium levels, one being only 2·1 mEq./l. in 3 of 4 neonates with intestinal obstruction, which suggests a deficit that should have been replaced.

Measurements of the specific gravity of the urine showed that the children, unlike adults, were able to dilute their urine shortly after operation. If this observation, which is in agreement with Calcagno, Rubin, and Singh's (1955) findings in infants, is confirmed in a larger series of children, the risks of relieving dehydration at the time of an operation are much less for children than for adults, who may pass into a phase of over-retention with circulatory difficulties and cerebral œdema (Le Quesne, 1957; Black, 1957).

Since confidence in the present plan of treatment has increased, it is now considered unnecessary to estimate the plasma electrolyte levels in most of the patients, but they are used as a valuable guide to the treatment of newborn infants, very sick children, and those with complications. Thus, soon after treatment is begun, low plasma sodium levels in a patient who has been profoundly dehydrated point to a relatively greater depletion of sodium than of water, and demand replacement with isotonic instead of hypotonic saline to avoid the risk of water intoxication. Low potassium levels, with hypochloræmia due to copious vomiting, should be treated by adding potassium chloride to the solutions for infusion from the outset. Later, a knowledge of the plasma electrolyte levels gives confidence to proceed with treatment. For instance, when the urine output has become scanty, as a result of inadequate replacement of gastro-intestinal losses, a normal or low plasma potassium level shows that potassium can be safely added at once to the solutions of sodium salts for replacement, despite the oliguria. Thus a progressive imbalance from giving sodium in excess without potassium is avoided.

Emergency operations must often be performed on dehydrated children, and their early fluid therapy can usually be planned without laboratory help, but complicated cases developing ileus should always be treated, by transfer if necessary, in a hospital with an emergency laboratory service. Elective operations for congenital intestinal abnormalities should be performed only in such places.

The post-operative care of infants and children with acute abdominal conditions demands meticulous attention to detail which can only be undertaken efficiently by a team of resident doctors, one of whom is always available to recognize the clinical indications for revising the course of therapy.

SUMMARY

The response to intravenous fluid therapy of children undergoing major abdominal surgery has been studied. The plan of treatment was designed to provide for relief of dehydration, if present, and for repair and maintenance of fluid and electrolyte equilibrium. Solutions of dextrose in varying concentrations of sodium chloride and lactate were used to relieve dehydration and restore urine flow. Thereafter, potassium salts were added to these solutions, so that intracellular as well as extracellular deficits were repaired, and further depletion from abnormal losses during the course of treatment avoided.

A consecutive series of 97 infants and children aged 1 month to 13 years and 23 newborn infants was treated according to this plan.

Five of each group died, in each case death being due to the severity of surgical disease, although two of the older children might have been helped by more intensive resuscitative treatment.

Clinical progress in response to intravenous fluid therapy was satisfactory in all but 4 of the 93 older children and in all the newborn infants who survived operation. These 4 developed convulsions due to water intoxication following incorrect treatment, the losses of gastro-intestinal fluid in these patients having been underestimated.

Intravenous fluid therapy was given for periods exceeding forty-eight hours in 22 patients in the older age-group, and in 17 of the newborn. The incidence of prolonged ileus was low (3 among 39 cases) after operations for inflammatory diseases, but was much higher after those for intestinal obstruction at all ages.

The urine flow was restored early in dehydrated patients following initial infusions given in amounts and at rates in accord with the schedule. Thereafter the urine was usually dilute and the volume rose as expected. The antidiuretic response which affects adults in the post-operative period was not apparent.

The plasma sodium levels were sometimes low (below 130 mEq./l.) in patients of both age-groups and occurred early and later in the course of treatment. They are regarded as an indication of salt depletion. The incidence in the older age-group has been correlated with failure to replace gastro-intestinal losses adequately.

The plasma potassium levels were sometimes low (below 3·8 mEq./l.) in patients of both age-groups and occurred early and later in the course of treatment.

The incidence of ileus was no higher among the patients with low, than among those with normal, levels at an early stage, and therefore, the hypopotassæmia is not regarded as the cause of the ileus. However, such patients would have been at risk from potassium deficiency if further losses had not been replaced. With the present method of treatment clinical effects of depletion were avoided.

Detailed studies of 3 children with ileus throughout the period of their intravenous fluid therapy have shown that 2 retained very large amounts of sodium and potassium, which are regarded as a measure of their losses at the outset. It is concluded that since such preceding losses are difficult to assess and further losses are unpredictable, a scheme of routine

treatment should include amounts of electrolytes to cover such large losses, as in the plan described here.

This plan of fluid therapy has proved satisfactory for children of all ages, and by its use the serious hazards from dehydration and from persistent ileus of the bowel can usually be avoided.

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CARCINOMA OF THE PARATHYROID GLAND

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THE present example of this relatively uncommon condition shows several interesting features; in particular, the parathyroid carcinoma had infiltrated and replaced one lobe of the thyroid. The neoplastic tissue closely resembled the normal thyroid gland, thus causing great difficulty in locating the tumour at operation.

CASE REPORT

A man aged 46 years was admitted to hospital in February, 1957, with recurrent hyperparathyroidism. The previous history was as follows:

In September, 1951, he was investigated at a hospital for dyspepsia of ten years' standing. The clinical history and X-ray findings were those of a duodenal ulcer. A partial gastrectomy was performed and a large stenosing ulcer was found in the first part of the duodenum.

In May, 1952, the patient complained of nausea, vomiting, anorexia, and loss of weight. A barium meal showed no evidence of ulcer or of dumping; a barium enema revealed generalized dilatation of the colon but no obstruction. As these gastric symptoms were distressing, the gastrectomy stoma was refashioned in July, 1953, but without relief of symptoms. Within a few months he was complaining, in addition, of tiredness and pain in the arms; he had a shuffling gait and was unable to lift his left leg. Increased frequency of micturition also occurred. These symptoms were so severe that he had to give up work.

A parathyroid tumour was suspected and the diagnosis was confirmed by the characteristic levels of serum

calcium, phosphorus, and alkaline phosphatase. In March, 1954, a soft encapsulated parathyroid tumour was removed from behind the left lobe of the thyroid. A second, smaller parathyroid gland was found on the same side of the neck; it was considered to be somewhat larger than normal.

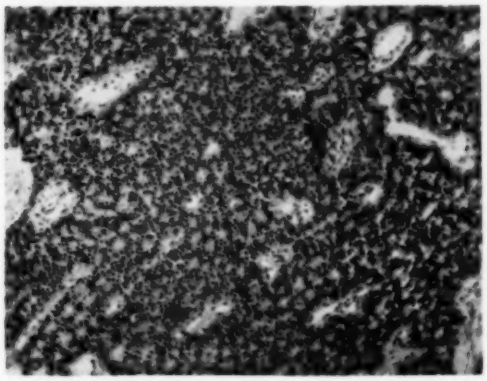

FIG. 280.—Tumour removed at first operation. The histology is that of a typical parathyroid adenoma composed of sheets of chief cells with a tendency to a perithelial arrangement. (× 90.)

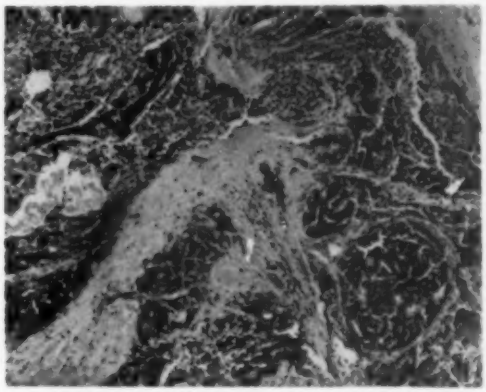

FIG. 281.—Another area from the same tumour as *Fig.* 280. Dense fibrous bands run between the sheets of epithelial cells. (× 64.)

Histological examination of the tumour showed that the greater part of it was composed of sheets of cells with prominent vascular spaces and a tendency for the cells to possess a perithelial or pseudo-acinar arrangement (*Fig.* 280). Occasional water-clear and eosinophil cells were present. No significant nuclear abnormalities were found, though prolonged search revealed a few mitotic figures. In some areas, however, the tumour cells were separated into small islets and columns of cells by dense bands of fibrous tissue (*Fig.* 281). Here the cells tended to be smaller and more darkly staining. Part of the periphery of the tumour was bounded by a thick fibrous capsule and was surrounded by slightly compressed thyroid tissue (*Fig.* 282). The specimen was originally reported as a benign parathyroid adenoma.

Troublesome post-operative tetany developed in spite of the administration of A.T.10 (dihydrotachysterol) and calcium lactate; soon afterwards the patient developed a subtrochanteric fracture of the right femur. After this episode he improved and six months later resumed work. However, in May, 1956 (26 months after the operation), his serum calcium was found at a routine check to be 14·5 mg. per 100 ml. During the next two months further examinations revealed persistently high calcium levels (15·6–16·7 mg. per 100 ml.) and low phosphorus levels (1·8–1·9 mg. per 100 ml.).

In August, 1956, the patient returned to his original hospital for re-exploration of the neck for a second parathyroid adenoma. No parathyroid tumour was discovered, but the entire right lobe of the thyroid was removed together with what seemed to be two enlarged parathyroid glands on that side. Histologically, one of the masses was an apparently normal parathyroid gland and the other was a foreign-body reaction round a suture from the previous operation.

No reduction in serum calcium or alkaline phosphatase levels followed, and the patient was admitted to Walton Hospital in February, 1957. His principal complaints at this time were generalized bone and joint pains and increasing weakness and vomiting. Polydipsia and polyuria were marked—he was passing 3 litres of urine a day. The blood-urea was normal, and though the left kidney shadow was poor the pyelogram revealed no gross abnormality. In particular, there was no visible renal calcification. In different examinations the serum calcium fluctuated between 17·4 and 14·8 mg. per 100 ml. and

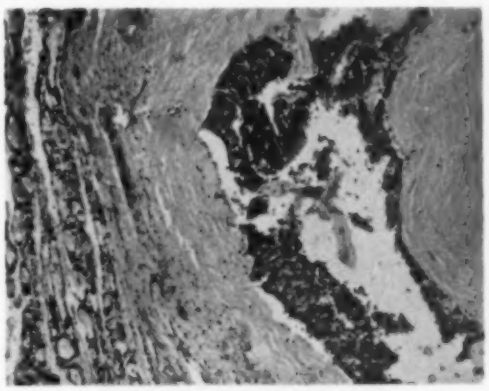

FIG. 282.—The edge of the tumour shown in *Fig.* 280. The surrounding thyroid tissue is rather compressed. (× 64.)

serum phosphorus between 2·2 and 2·5 mg. per 100 ml., with a serum alkaline phosphatase averaging 60 units per 100 ml. Radiographs showed further progression of skeletal decalcification.

A serious therapeutic dilemma was now present. From the reports from the previous hospital it seemed probable that only a single parathyroid gland remained and that this was the likely site of the tumour. If so, removal of the tumour would be expected to produce permanent hypoparathyroidism. However, it was decided to accept this risk, and in May, 1957, the third exploration of the neck was undertaken (P. R. H.). A tumour 15 × 10 × 5 mm. in size was found in the superior mediastinum behind the clavicle on the left side; frozen sections showed that it was a chief-cell adenoma of the parathyroid, with a peritheliomatous arrangement and pseudo-acini exactly resembling the more cellular areas of the tumour removed at the first operation. A thin fibrous capsule was present, but there were no dense fibrous bands. No lymphoid tissue was seen. The neck was carefully explored, both on the right and on the left

sides, but no further tumours were seen. A small piece of the parathyroid tumour was inserted into the rectus sheath as a precautionary measure against hypoparathyroidism.

Post-operatively, a saline drip containing calcium gluconate was set up and A.T.10 prescribed as a prophylactic measure. On the first five post-operative days the serum calcium levels were all elevated, ranging from 14·4 to 18·1 mg. per 100 ml. despite stopping the calcium gluconate drip on the second post-operative day. It was considered possible that the persistently high levels were due either to the A.T.10, of which 25 ml. had been administered in 5 days, or to the tumour autograft. The A.T.10 was discontinued, and on the eighth day the graft was removed, but no reduction in serum calcium resulted. Histologically, the graft was alive. It consisted of a mass of granulation tissue with some foreign-body giant cell reaction to suture material. In the superficial parts of this granulation tissue there were islets and cords of live epithelial cells with pale or rather eosinophil cytoplasm, which were clearly parathyroid cells.

From this time the patient suffered from persistent vomiting and increasing weakness. His serum calcium remained between 15·3 and 18 mg. per 100 ml., and it was clear that hyperfunctioning parathyroid tissue remained. Three weeks after the previous operation the neck was explored for the fourth time. An attempt was made to locate the suspected tumour by the use of diodone injection into the inferior thyroid artery as described by Steiner, Fraser, and Aird (1956), but in the present case this vessel was very small, probably as a result of the previous operations, and no useful radiological information was obtained. The mediastinum was examined widely after splitting the sternum, but no tumour could be found and the neck was therefore re-explored. The remaining small portion of the upper lobe of the thyroid gland was incised and a small round mass 1·5 mm. in diameter was found in its substance; frozen sections showed that it was a thyroid adenoma. Further search revealed a fibrous mass attaching the remnant of the left lateral lobe of the thyroid to the trachea and cricoid cartilages. This was thought to be scar tissue resulting from the previous operations, but a piece was examined in frozen section and found to be composed of dense bands of fibrous tissue enclosing islets of rather dark parathyroid cells, closely resembling the scirrhous areas in the tumour removed at the first operation.

By this time the condition of the patient would not permit any further exploration and the operation was terminated. Throughout the final stages of the operation general oozing from the exposed tissue was a serious problem and could not be adequately controlled. Death occurred four hours after operation.

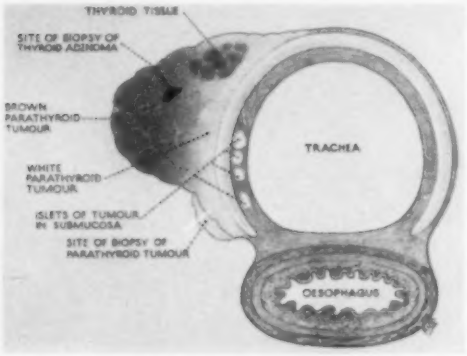

FIG. 283.—Post-mortem appearance of the tumour and adjacent structures.

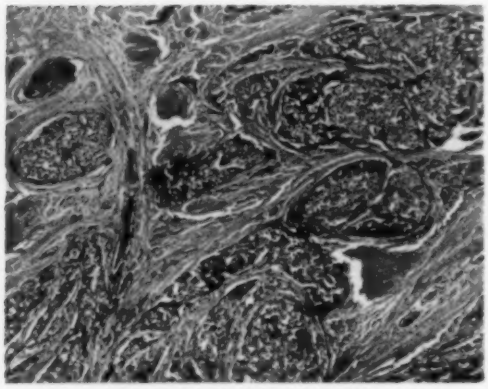

FIG. 284.—Parathyroid tumour replacing lobe of thyroid. (× 64.)

FIG. 285.—Parathyroid tumour infiltrating tracheal wall. (× 64.)

POST-MORTEM FINDINGS.—The post-mortem examination was performed two hours after death.

Thyroid and Parathyroid.—The right lobe of the thyroid and the lower part of the left lobe had been removed previously. The remainder of the left lobe was represented by a rounded mass about 3 × 4 × 2 cm., with a fresh incision in its anterior surface, through which the thyroid adenoma had been removed at the fourth operation (*Fig.* 283). At the back of the left lobe, at the site where the biopsy had shown scirrhous parathyroid tumour, there was a thin plaque of brownish tissue firmly adherent to the wall of the trachea over the upper two or three rings.

Histologically, the 'left lobe' was found to be composed, not of thyroid gland, but of parathyroid tumour. Only a small mass of thyroid tissue remained, about 1 cm. in diameter (*Fig.* 283), and the rest of the lobe was replaced by a rather scirrhous parathyroid tumour. The part nearest the trachea was firm and white, but farther out it was softer and more brown, so that macroscopically it closely resembled thyroid tissue. The tumour had penetrated between the cartilages of the trachea and was growing as isolated nodules about 1–2 mm. in diameter in the submucosa of the trachea.

Histologically the tumour showed the same picture as the scirrhous areas from the tissue removed at the

first operation (*Figs.* 284, 285); sheets of chief cells with a remarkably uniform appearance were separated by broad strands of dense fibrous tissue. Rare mitoses were found. No other parathyroid tissue could be found in the neck or mediastinum.

Bones.—All the bones were soft and easy to cut. Large fleshy osteoclastoma-like masses were present in the ribs and skull (*Fig.* 286). Histologically, the bones showed the typical appearance of osteitis fibrosa.

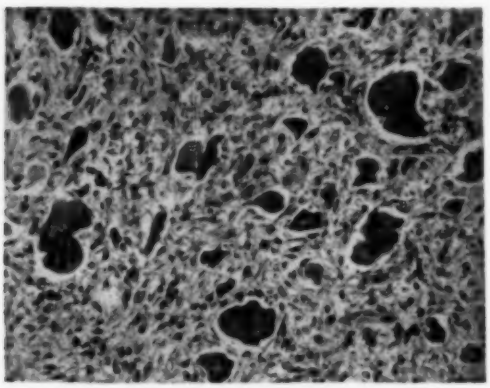

FIG. 286.—Osteoclastoma-like mass from rib. (× 135.)

Other Organs.—The upper posterior pleura on both sides of the chest showed numerous hard white button-like deposits of calcium salts, ranging from about 1–10 mm. in diameter.

The left lung had a layer of gelatinous fibrin on the pleural surface, but otherwise the lungs were normal.

The heart showed numerous subendocardial hæmorrhages of the type seen in shock.

The stomach had been subjected some years ago to partial gastrectomy. No active ulcer was present.

The kidneys both weighed 165 g. On naked-eye examination there were small whitish flecks in the deep cortex, and many white streaks were seen in the pyramids. Microscopically, the cortex showed patches of interstitial fibrosis, with atrophy of tubules, hyalinization of glomeruli, and some lymphocytic infiltration. Small foci of calcification were common. Some were in the interstitial tissue, and others were in the tubules, where they often excited a reaction of the epithelial cells, which partly covered and penetrated the granular mass of calcium salts. These findings agree with the description given by Young and Emerson (1949). No calculi were found in the urinary tract.

The other organs were normal; in particular, a careful search showed no islet-cell adenomas or pituitary adenomas.

DISCUSSION

Pathology of the Tumour.—Proved cases of parathyroid carcinoma are uncommon; the reported cases are discussed by Norris (1948), Leger, Lièvre, and Lièvre (1953), and Black (1954).

As has been suggested by King and Wood (1950) and Wray (1953), there are two criteria which must be fulfilled to establish the diagnosis of parathyroid carcinoma.

1. There must be functional evidence that the tumour arises from parathyroid tissue. This is necessary in view of the fact that thyroid carcinoma can mimic parathyroid tumours histologically. In the present case the clinical history, the radiological findings, the serum chemistry, and the post-mortem appearances in the bones provide conclusive evidence that the patient had suffered from an excess of parathyroid hormone. The systemic findings in this case presented no unusual features and do not require a detailed discussion; attention may, however, be drawn to the excellent account of the morbid anatomy in hyperparathyroidism with a review of the early literature, given by Hoffheintz (1925).

2. Infiltration or metastasis must be present before malignancy can be diagnosed. Purely histological criteria, such as nuclear irregularity, which have been used by certain authors (in particular by Alexander, Pemberton, Kepler, and Broders, 1944), are not acceptable. In the present case the infiltration of the thyroid gland and of the trachea is the evidence for malignancy. No definite metastases were found; the parathyroid tumour removed at the third operation had all the appearances of a separate adenoma, although it might be argued that it represented the complete replacement of a lymph-node by secondary tumour. This latter interpretation is supported by the case reported by de Wesselow and de Wardener (1949), who found multiple nodules of 'parathyroid adenoma' in the neck of a woman with carcinoma of the parathyroid. Albertini, Koller, and Gaiser (1953) also record the removal of an 'adenoma' from the neck of a patient with parathyroid carcinoma.

The epithelial cells in the present tumour showed no more irregularity in size and nuclear type than is found in benign adenomas, and this agrees with the accounts by previous authors (Ellis and Barr, 1951; O'Donovan, Meade, and McGrath, 1951; Albertini and others, 1953; Wray, 1953). However, in a case described by Black and Ackerman (1950) with a later report by Black (1954), there were 'bizarre' chief cells with little variation in nuclear size, and Black (1954) also reports 2 further cases with areas of cell pleomorphism. In the present case mitoses were present, though very scanty. Castleman (1952) believes that mitoses, however rare, in a parathyroid tumour are indicative of carcinoma. Leger and others (1953) found atypical mitoses in their case of parathyroid carcinoma.

A conspicuous feature was the presence of dense fibrous tissue bands. As has been suggested by Cope, Nardi, and Castleman (1953) this would appear to be a better criterion of malignancy than the character of the tumour cells themselves, for a similar appearance has been recorded several times in the accounts of the histology of parathyroid carcinomas (Young and Emerson, 1949; de Wesselow and de Wardener, 1949; Black and Ackerman, 1950; King and Wood, 1950; Cope and others, 1953; O'Donovan and others, 1951).

Perhaps the chief interest of this case was that nearly all the remaining left lobe of the thyroid was replaced by parathyroid tumour. Since the colour of the tumour was indistinguishable from the colour of normal thyroid tissue, this caused great difficulty at the time of operation. Such replacement and mimicking of the thyroid by parathyroid tumour does not appear to have been described before, although several authors have mentioned infiltration of the thyroid by parathyroid carcinoma (Gutman, quoted by Albright and Reifenstein, 1948; O'Donovan and

others, 1951; Wray, 1953). Since a normal parathyroid gland is occasionally situated within the substance of the thyroid gland (Gilmour, 1938) and about 1–3 per cent of parathyroid adenomata are intrathyroid (Norris, 1948; Black and Haynes, 1949; Lahey and Murphy, 1953; Hellström, 1954), it is clear that a careful examination of the thyroid itself should be made when searching for parathyroid tumours. Hellström, indeed, believes that a subtotal thyroidectomy is justified if a thorough exploration of the neck and mediastinum fails to reveal a parathyroid tumour.

Result of the Autograft Operation.—The fact that live parathyroid cells were present in the autografted tissue when it was removed eight days after operation indicates that the graft had taken satisfactorily, and, had it been left, it might have secreted hormone. As has been discussed above, it is not certain whether the tumour from which the graft was taken was an independent adenoma or a metastasis from the main carcinoma.

The survival of autographed parathyroid tissue has been reported by other workers. Goldman (1955) found live chief and oxyphil cells in an autograft nine days after transplantation, and in the case described by Burk (1947) the graft grew. Although in the present case subsequent events proved that the graft had not been necessary, the method does seem to have a possible value in cases where permanent post-operative hypoparathyroidism is anticipated.

Dihydrotachysterol.—The administration of dihydrotachysterol after the third operation raised the problem of the precise time-relationships of its action. Most standard text-books of pharmacology stress the cumulative effect of dihydrotachysterol. In the present case, large doses were given to prevent an anticipated tetany and, when the post-operative calcium levels remained high, consideration was given to the possibility that the hypercalcæmia was due to the action of the drug. Albright, Bloomberg, Drake, and Sulkowitch (1938) showed that dihydrotachysterol has a somewhat similar action to that of vitamin D; it produces a positive calcium balance and a negative phosphorus balance, and acts more rapidly than vitamin D. Albright and others found that the serum calcium rose before the third day after beginning treatment and began to fall three days after the administration of the drug ceased. McChesney and Messer (1942), working with dogs, also stressed that dihydrotachysterol causes a more rapid rise and fall of serum calcium than does vitamin D. Persistent elevation of serum calcium over a long period after cessation of dihydrotachysterol is therefore unlikely to be due to the drug, and another cause must be sought.

Association with Peptic Ulcer.—It seems probable that the occurrence of a gastric ulcer in this patient was not a mere coincidence, since there are now many cases reported in which peptic ulcer was found in association with hyperparathyroidism (Rogers, 1946; Rogers, Keating, Morlock, and Barker, 1947; Robinson, Black, Sprague, and Tillisch, 1951; Black, 1953; Lahey and Murphy, 1953; Hellström, 1954; Tsumori, Jensen, Hunnicutt, Foremen, and Kinsell, 1955; Crawford, Steffanelli, and Alvarez, 1956). The ulcer in most cases has been situated in the duodenum, but Elkeles (1953) reports the case of a woman with hyperparathyroidism and both gastric and duodenal ulcers. Hellström in his series found that 14 per cent of cases of hyperparathyroidism had ulcer, and Black (1953) claimed an incidence of 24 per cent. If the incidence is as high as these authors suggest, it would seem reasonable to perform at least a Sulkowitch test for urinary calcium as a routine in all cases of peptic ulcer. The occurrence of vomiting in cases of gastric or duodenal ulcer in the absence of stenotic lesions should perhaps particularly be regarded with suspicion.

A curious feature in the present case is the long interval that elapsed between the onset of the peptic ulcer and the development of overt hyperparathyroidism. Several of the reports listed above (e.g., Rogers; Rogers and others; Robinson and others, Hellström) also describe cases in which peptic ulcers developed many years before the parathyroid adenoma was diagnosed.

SUMMARY

A case is described of a carcinoma of the parathyroid in a 46-year-old man. Symptoms definitely referable to the hyperparathyroidism developed five years before death. The radiological changes in the bones and the serum chemistry findings were typical, and a remission was produced by the surgical removal of a tumour. The symptoms recurred, however, and persisted despite three further surgical interventions.

An interesting feature is that the parathyroid carcinoma had infiltrated and largely replaced one lobe of the thyroid gland. Since the tumour was brown in colour, it very closely mimicked the thyroid tissue on naked-eye examination, and so its true nature was very difficult to recognize at the time of operation.

Histologically, the tumour was characterized by a dense fibrous stroma; this seems to be a fairly constant feature of malignant parathyroid growths.

As in many previously reported cases of hyperparathyroidism, the patient also suffered from a peptic ulcer; this preceded the onset of definite symptoms of hyperparathyroidism by about eleven years.

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APPENDICULAR CALCULI

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TRUE calculi in the appendix are rarely encountered (Bunch and Adcock, 1939), though radio-opaque fæcoliths would appear to be much less uncommon (Steinert, Hareide, and Christiansen, 1943). Neither condition has received much attention from British authors, and inquiry among surgical and radiological colleagues suggests that their occurrence is not widely recognized.

The following case well illustrates many of the typical features of appendicular calculi, and was of unusual interest from the diagnostic standpoint.

FIG. 287.—Radiograph showing faceted calculi in the line of the right pelvic ureter.

FIG. 288.—Radiograph showing gross gaseous distension of the small intestine. The calculi are now lying transversely.

CASE REPORT

Mrs. R. E., aged 47 years, was admitted to hospital as an emergency case on Jan. 12, 1956. One month previously she had suffered an attack of severe colicky suprapubic and right-sided abdominal pain lasting about

twelve hours. There had been associated vomiting and also some frequency and pain on micturition. Her doctor suspected ureteric colic and treated her at home. A subsequent straight radiograph of the abdomen (*Fig.* 287) showed three laminated and faceted shadows in the right side of the pelvis, lying in the line of the right ureter.

Two days before admission the patient developed a further attack of colicky right-sided abdominal pain, with accompanying anorexia, constipation, and a feeling of

FIG. 289.—Photograph of the appendicular calculi.

distension. She also had some dysuria, and her doctor reported slight hæmaturia. On this occasion the symptoms persisted and she was sent into hospital on Jan. 12.

ON EXAMINATION.—The patient was in good general condition, but considerably distressed by her pain. Temperature was 97° F., pulse-rate 110 per minute. The abdomen was slightly distended, and there was tenderness and guarding in the right iliac fossa and in the right loin. Rectal examination revealed tenderness on the right side of the pelvis. The urine contained a trace of albumin, but no macroscopic blood. There were no other significant findings.

A provisional diagnosis of right ureteric colic was again made, as this seemed to fit in with both clinical and radiological findings. The author was not at the time familiar with the occurrence of appendicular calculi, and the correct diagnosis was not considered.

PROGRESS.—The patient's pain persisted and on the following day she became pyrexial. By Jan. 14 the pain had extended to the left iliac fossa. Examination showed the abdominal distension to have increased considerably, and tenderness and guarding were now more diffuse. A further straight radiograph (*Fig.* 288) revealed that the three faceted calculi were now lying transversely in the right side of the pelvis, and several greatly distended coils of small bowel were present. The erect film showed multiple fluid levels. The radiologist reported: "There appear to be three faceted gall-stones in the right side of the pelvis". A diagnosis of acute intestinal obstruction was made, and immediate laparotomy advised.

AT OPERATION (Jan. 14).—The abdomen was opened through a right paramedian incision. The lower ileum was found to be obstructed by fibrinous adhesions in the wall of a pelvic abscess. The cause of the latter was a perforated appendix containing the three calculi previously seen on the radiographs. Appendicectomy was performed, the obstruction freed, and the abdomen closed with drainage. Post-operative progress was uneventful, apart from the development of a late wound infection necessitating the patient's readmission to hospital twenty-six days after operation.

SPECIMEN.—The whole appendix showed severe acute inflammation with gangrene and perforation. On slitting up the organ four small diverticula were seen on the mesenteric aspect. Lying free in the lumen were three hard faceted calculi, whitish in colour, each about 1 cm. in diameter (*Fig.* 289). On section, the calculi were found to have a hard outer shell some 3 mm. thick, with a softer, rather flaky, brown central core. Chemical analysis showed the stones to contain calcium, phosphate, and a small amount of carbonate. The centres contained a small quantity of organic matter.

DISCUSSION

Incidence.—There is considerable apparent divergence of opinion regarding the frequency of appendicular calculi. Thus Bunch and Adcock (1939) found only one example in over 2000 cases of acute appendicitis, and inquiry among surgical colleagues supports my own impression of the rarity of the condition. On the other hand, Brady and Carroll (1957) found 34 cases in one 5-year period at the John Gaston Hospital, Memphis, Tennessee. These apparent discrepancies are doubtless to be explained by differences in terminology. If by the term calculus one implies a concretion with the appearance and hardness of a stone, then appendicular calculi are rare. Inclusion of all concretions with sufficient calcification to show up on a plain abdominal radiograph would make the condition relatively common. Thus Steinert and others (1943) found calcified fæcoliths on routine radiological examination in 10 out of 104 cases of appendicitis, while Felson (1949) found radio-opaque 'calculi' in 25 out of 75 acutely inflamed appendices radiographed after removal. He estimated that in 9 of these the shadow would probably have been sufficiently dense to show up on a pre-operative radiograph had one been taken. It is apparent that many of these radio-opaque appendicular concretions are merely fæcoliths containing considerable amounts of calcium phosphate, yet remaining soft and showing little macroscopically to distinguish them from the common radio-translucent fæcolith.

In the present case the calculi were stony hard, whitish in colour, and bore no resemblance to fæcoliths. They may be classified as true calculi.

True calculi and calcified fæcoliths have similar radiological features, and the remarks that follow apply in the main to both groups.

Aetiology.—It is thought that as a result of stasis fæcal material in the appendix becomes inspissated and forms one or more fæcoliths. These cause irritation of the mucosa with resultant increase in mucus secretion, and inorganic salts, particularly calcium phosphate, are then precipitated around the periphery of the fæcolith. Foreign bodies or even small gall-stones may occasionally enter the appendix and act as a nidus around which a calculus forms.

Appendicular stasis would therefore seem to be a predisposing factor. In the present case the presence of multiple appendicular diverticula in addition to the calculi was interesting in the light of Wilson's (1950) finding that an element of appendicular obstruction is necessary for the development of diverticula.

Composition.—The mineral content of appendicular calculi is nearly always predominantly calcium phosphate, and this was so in the present case. The average composition is given by Felson and Bernard (1947) as calcium 20 per cent, phosphorus 10 per cent, and coprosterol 10 per cent.

Diagnosis.—The pre-operative diagnosis of appendicular calculi rests on the correct interpretation of a shadow or shadows on an abdominal radiograph. Very few calculi are discovered as incidental findings in patients having radiographs taken for other purposes, for in over 90 per cent of reported cases they have been radiologically demonstrated for the first time during an attack of acute appendicitis. The attack is usually a severe one, and the incidence of perforation in these cases is high, e.g., 32 out of 34 cases in Brady and Carroll's (1957) series. Two factors are of causal importance here. The first and most obvious is that a calculus will tend to cause appendicular obstruction at an early stage of an attack of appendicitis. The second factor is that in many of the reported cases the clinical features have been somewhat atypical of appendicitis. Had this not been so the patients would not have been subjected to radiological examination, except, as in Steinert and others' (1943) series, as part of a deliberate research study. Delay in advising operation is more likely to occur in the clinically atypical case, and furthermore, a calculus seen on the radiograph may be mistaken for some other condition with possibly further delay. This happened in the present case, and it is probable that had the patient not had the 'advantage' of an X-ray at all, the appendix would have been removed forty-eight hours earlier.

Fortunately the radiological features of appendicular calculi are often sufficiently characteristic for the correct diagnosis at least to be suggested. They are more often single than multiple; if multiple they may be faceted, as in the present case, though the demonstration of facets may be difficult (Felson and Bernard, 1947). The shadow(s) may be seen in any position that the appendix can occupy, most commonly the right iliac fossa or right side of the pelvis. The most reliable single feature is peripheral lamination.

Calculi in the right ureter provide the most likely source of confusion, as in the present case and in cases described by Sawyers and Rosenfeld (1953) and Prather and Singiser (1953). However, ureteric calculi rarely show well-marked peripheral lamination. In a doubtful case further radiographs after the passage of a ureteric catheter should be done without delay.

Gall-stones have also been simulated when either the gall-bladder or appendix has been in an unusual position, or when it has been thought that a gall-stone has migrated to the small bowel and become impacted in the lower ileum (Chapple, 1951). Gall-stone ileus was simulated in the second radiograph (*see Fig.* 288) in the present case, and in a case described by Moloney (1947).

Radiological features should serve to exclude other types of shadows in the right lower quadrant, e.g., calcified nodes, phleboliths, bone islands in the ileum, calcification in the iliac arteries, and foreign bodies.

Treatment.—The majority of patients in whom appendicular calculi are radiologically demonstrated will be suffering an attack of acute appendicitis, and the treatment differs in no way from that of acute appendicitis in general, save that at operation in perforated cases a search should be made in the neighbourhood for the calculus, if the latter has escaped from the appendix.

In the rare event of the incidental radiological discovery of a suspected appendicular calculus, and its verification if necessary by barium and other special studies, all authors are agreed that early appendicectomy should be advised, because the risk of obstructive appendicitis subsequently developing is great.

SUMMARY

A case of multiple appendicular calculi is described. The appendix also presented several diverticula. Acute appendicitis supervened with clinical and radiological simulation of ureteric calculi, and later of gall-stone ileus. The incidence, aetiology, diagnosis, and treatment of appendicular calculi are discussed.

I wish to express my thanks to Mr. R. H. Franklin for permission to publish this case, and to the Photographic and Biochemical Departments, Westminster Hospital, for their help.

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THE PROPHYLAXIS OF PULMONARY EMBOLISM

A CLINICAL PROGRAMME

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THE problem of pulmonary embolism remains formidable in spite of great advances in medicine and surgery. Some clinicians especially interested in this condition record some progress. Thus Allen (1946) reports a reduction in the incidence of fatal pulmonary embolism from 3 in 1000 to 1 in 800, by careful attention to the prevention of venous stasis. Gjores (1954) obtained a history of venous thrombosis in 183 of 8560 of the general population in certain districts in Sweden. Gibbs (1957) found that 39 cases (13 per cent) in a total of 219 cases (average age 61 years) showed old thromboses in the veins of the lower limbs at necropsy which were unrelated to the last fatal illness. These findings give some indication of the extent of the problem.

The necropsy dissections of the morbid anatomists (Rossle, 1937; Neumann, 1938; Putzer, 1939; Frykholm, 1939, 1940; Hunter, Sneeden, Robertson, and Synder, 1941; Hunter, Krygier, Kennedy, and

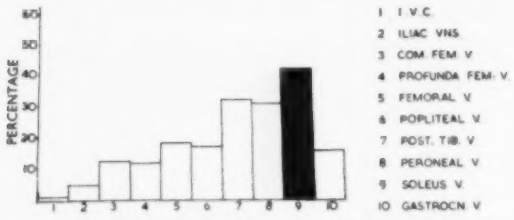

FIG. 290.—Distribution of thrombi within the veins of 476 lower limbs (239 cases). Note that the greatest incidence of thrombosis occurs in the intramuscular veins of the soleus muscle.

Sneeden, 1945; Greenstein, 1945; Raeburn, 1951; McLachlin and Paterson, 1951; Gibbs, 1957) have demonstrated the high incidence of venous thrombosis of the lower limbs in patients confined to bed in hospital. In the majority, the thromboses were 'silent' and not often manifest by clinical signs and symptoms during life. The failure of the clinicians to appreciate that massive pulmonary embolism was a sequel of only a small proportion of 'silent' venous thromboses had led to much confused thinking. The relationship between venous thrombosis of the lower limbs and the duration of bed-rest has been fully investigated by Gibbs (1957).

APPLIED ANATOMY OF VENOUS THROMBOSIS

It has been demonstrated by Gibbs that venous thrombosis of the lower limbs occurs in two quite separate and clearly defined sites, namely, the leg and the thigh.

Thrombosis of Leg Veins.—Thrombosis is most common in the intramuscular veins of the soleus muscle (*Fig.* 290). This is due to the wear and tear inflicted upon these veins as a result of the unique function of the muscle which acts as a venous pump returning blood from the limb against gravity. It has been shown by Gibbs that these veins undergo progressive distortion and dilatation with advancing age, and this largely explains the predilection of venous thrombosis of the lower limbs for adults over the age of 40 years. These veins are dependent when

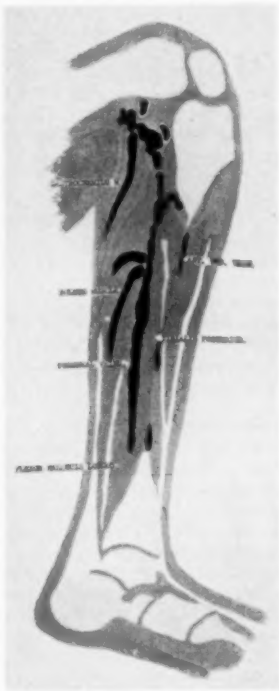

FIG. 291.—Venous anatomy of the leg. Soleus veins are dependent when the limb is supine.

the lower limb is supine (*Fig.* 291) and venous blood tends to pool in conditions of muscular inactivity and immobility. It has been shown that the venous endothelium is very susceptible to anoxia (O'Neill, 1947; Impallomeni, 1954), which may develop in conditions of venous stasis. Damage to valve cusps by apposition of intimal surfaces is an additional cause of endothelial injury.

Thrombosis of Thigh Veins.—Thrombosis in the veins of the thigh occurs near the inguinal ligament (*Fig.* 292) where many large veins meet to form the common femoral vein. At this site venous

stasis is very liable to occur. The femoral venous flow is largely dependent upon the muscular contraction of the soleus muscle and therefore venous stasis at the inguinal ligament is aggravated by stasis in the veins of the leg.

VENOUS THROMBOSIS DURING BED-REST

Necropsy evidence shows that calf-vein thrombosis may begin after a period of three days, and thigh-vein thrombosis within seven days, of confinement to bed, while after one week venous thrombosis

FIG. 292.—The veins of the left thigh. This is the site where venous stasis is particularly liable to occur in the veins of the thigh.

is commonplace. The incidence of venous thrombosis, thigh-vein thrombosis, and pulmonary embolism in a series of 239 patients confined to bed for a maximum period of twelve weeks before death is shown in *Fig.* 293 (Gibbs, 1957). Thigh-vein thrombosis is generally more dangerous than leg-vein thrombosis, since thrombi detached from the former are larger and produce more effective occlusion of the lumen of the pulmonary artery. The problem is to find some means whereby venous stasis can be overcome at an early stage in the post-operative period or prevented altogether in medical patients. The efforts of the physiotherapists have failed for a number of reasons. These include lack of understanding concerning the mechanics of venous stasis, whereby attention has not been directed towards the most important muscle, namely the soleus muscle. It has been proved impossible to instruct patients to perform adequate active movements during the waking hours while confined to bed and not under the direct supervision of the physiotherapist. The phrase 'keep the toes moving' is often the only contribution made by the clinician in the prophylaxis of venous stasis. Movement of the toes does not exercise the soleus muscle. Raising the foot of the bed fails to provide dependent drainage for the soleus veins unless the angle of elevation is at least 60°. The state of affairs in ambulant patients is also unsatisfactory. Early ambulation has only been partially successful as a prophylactic measure (Blodgett and Beattie, 1946; Canavarro, 1946; Powers, 1947). Early rising in the post-operative period, particularly in middle-aged patients who have had extensive abdominal operations, is an exhausting procedure. The active exercise attained by a few faltering steps from the bedside is paid for by hours of immobility in bed while the patient recovers from the pain thus caused. As ambulation progresses patients are herded into arm-chairs so that the centre of the ward may provide a 'clear run' for the medical and nursing staffs, and there is no doubt that the design of many hospital wards and corridors is unsuitable for the practice of adequate ambulation. An ambulant patient should spend the time out of bed either standing or walking,

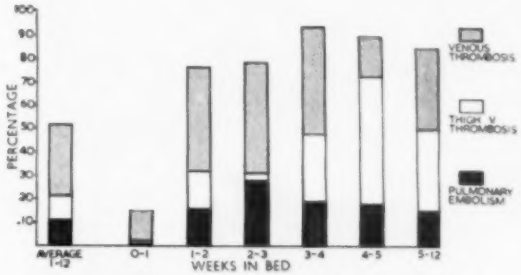

FIG. 293.—Incidence of venous thrombosis and pulmonary embolism (0–12 weeks' bed-rest).

and prolonged recumbency in arm-chairs is even more dangerous than bed-rest, since the risks of venous stasis are greater. The accent by the senior nursing staff on pernickety tidiness, and the silent inactivity demanded by the medical and surgical staffs during ward rounds, which in some teaching hospitals may succeed one another for the greater part of the day, aggravate the problem.

THE PROPHYLAXIS OF VENOUS STASIS DURING BED-REST

The development of venous stasis is a necessary preliminary to the formation of the venous thrombus, and once this has occurred the patient is in danger and the train of events which may lead to pulmonary embolism is set in motion. The prophylaxis of venous stasis in the lower limbs is dependent, firstly, upon attaining adequate exercise of the soleus muscles and thigh muscles, and secondly, on preventing venous stasis and consequent turbulence of blood-flow in the thigh veins near the inguinal ligament. These two main factors, although partly interdependent, will be considered separately.

Venous Stasis of the Leg.—Adequate exercise of the soleus muscle must be carried out during the waking hours. To ensure that this is done some method must be found whereby:—

1. The soleus muscles are required to contract against an adequate load for a definite time.
2. This load should be calculated depending upon the muscular build and clinical condition of the patient.
3. Some system should be instituted whereby an accurate check can be made of the exercise actually performed by the patient.
4. The calf muscles should be elevated from the surface of the mattress during the performance of the exercises, so that contraction of the soleus muscle

should be facilitated and compression of the intramuscular veins avoided.

5. The above four requirements should be made possible without greatly increasing the work of the nursing or physiotherapy staffs and without great expense.

To this end, the following piece of apparatus has been designed.

The Soleus Ergometer (*Figs.* 294–296) consists of a vertically mounted, rubber-padded platform with a curved heel support. The platform is pivoted at the base, so that the platform may rotate through an angle of 25° when the foot is plantar flexed. The midpoint of the platform is fixed to a spring with adjustable tension which is compressed during active plantar flexion of the feet. Each complete traverse of the platform produced by plantar flexion is recorded on a meter. If the traverse is incomplete the meter does not record. The instrument is mounted on an adjustable carriage which can be fixed to the bed-frame. The position of the ergometer in the bed can thus be readily adjusted. A curved hoop is attached to the carriage to raise the bedclothes and allow free use of the apparatus. When the instrument is not in active use it does not impede free movement of the lower limbs in bed.

FIG. 294.—Soleus ergometer, showing construction of the apparatus.

FIG. 295.—Soleus ergometer, showing apparatus mounted on the bed-frame and feet in the resting position.

The Soleus ergometer records full contractions of the soleus muscle against an adjustable resistance. This is accompanied by synergic contraction of the dorsiflexor muscles of the leg and the extensor and flexor muscles of the knee-joint. The ergometer fails to record if the platform is not rotated through the full arc, that is, if the movement of plantar flexion of the foot is incomplete. By this means the accurate control of exercise performed by the soleus and thigh muscles, which are responsible for the venous return from the lower limbs, may be achieved.

Use of the Ergometer.—The spring tension and position of the instrument in the bed may be adjusted with regard to height, age, strength, and muscular development of the patient. A minimum average of thirty recorded acts of plantar flexion per hour during the waking hours is thought to be adequate. Generally, each surgical patient, for the 48-hour period following operation, will require exclusive use of an ergometer. Similarly, bad-risk cases should have exclusive use of the apparatus until full ambulation is attained. For other patients, the waking hours may be subdivided into periods of four hours, so that each patient may have exclusive use of the ergometer for one hour in every four hours, during which time a minimum of 120 contractions should be recorded. It has been found that patients who have had extensive abdominal operations, e.g., gastrectomy, can use the ergometer without difficulty within forty-eight hours of operation.

FIG. 296.—Soleus ergometer, showing apparatus in use.

Venous Stasis of the Thigh.—Venous stasis of the thigh occurs partly as a secondary effect due to inadequate venous return from the leg muscles, particularly the soleus muscle, and partly due to anatomical factors existing at the inguinal ligament, whereby turbulence of the venous stream occurs. A successful solution to the problem of venous stasis within the veins of the calf, and particularly the soleus muscle, will in itself reduce the probability of thigh-vein thrombosis. However, special consideration should be given to the thigh veins near the

inguinal ligament. It has been demonstrated (Gibbs, 1957) that configuration of the common femoral vein is readily distorted by the inguinal ligament, and particularly by pronounced flexion and abduction. Prolonged periods in the Fowler position should be avoided, particularly in obese patients, but the essential factor is the frequent changing of position whereby turbulence of the venous stream at the inguinal ligament is minimized.

CLINICAL PROGRAMME

The basic principles have been outlined whereby the basal activity of the muscles of the lower limbs is increased and the 'trough of inertia' (Gibbs) which follows bed-rest is prevented. The following clinical programme has been devised for surgical and medical patients who enter hospital from waiting lists, the majority of whom are ambulant prior to admission. It is recognized that some patients may be admitted who have been confined to bed at home and some modifications to this programme, which will be discussed, are required.

Surgical Cases.—

General Considerations.—The advancements in anæsthetic techniques and blood transfusion have enabled larger and more complicated surgical procedures to be undertaken which in themselves promote venous stasis. It is important that the patient should regain consciousness as soon after the operation as possible and post-operative sedatives be given with caution. It must be emphasized that 'cold' surgical operations undertaken on patients who have been confined in hospital for more than a week are most dangerous. Where possible, patients requiring operation under these circumstances should be sent home for a month, so that full activity can be restored prior to operation. Furthermore, this period will allow time for organization of any thrombi that may have been formed during the period of investigation in hospital. On admission, the patient should be instructed by a member of the staff, usually the houseman, on the use of the Soleus ergometer, and the apparatus should be adjusted for position and spring tension. This apparatus should be used by the patient during the waking hours from the time of confinement to bed. It should be explained that co-operation by the patient can be checked by means of the trip recorder. The recorder reading is then charted with the temperature, pulse, and respiration.

Post-operative Period.—Immediately the patient is conscious and can co-operate the apparatus should be used, the lower limbs being placed in position with the help of the nursing staff, if necessary. Henceforth, soleus muscle exercise should be charted 4-hourly with the temperature, pulse, and respiration for the usual 24 hours. After 24 hours, the readings of the recorder may be noted routinely with the temperature, pulse, and respiration. Special attention may be necessary in those patients who fail to maintain adequate basal exercise, and the recording apparatus will therefore direct the attention of the medical and nursing staffs where it is most needed. It is most important that the position of the recumbent patient should be changed every two hours to reduce the effects of turbulence in the veins near the inguinal ligament.

Ambulant Period.—Adequate use of this apparatus will enable the lower limb muscles to retain their power and will aid in the preparation of the patient for ambulation. Nevertheless, the apparatus should be used by patients when they retire to bed. It is much safer for patients to rest in bed and use this apparatus than to spend long periods sitting in arm-chairs. When confined to bed during the waking hours, the position should be changed at least every two hours.

Medical Cases.—

General Considerations.—The problem of preventing venous stasis in medical cases is generally less difficult, and adequate use of the Soleus ergometer should avert venous thrombosis. It is recognized that disability suffered by some patients may preclude sufficient use of the apparatus. The reading of the recorder should be noted routinely with the temperature, pulse, and respiration, and patients who fail to undertake adequate exercise should be singled out for special attention by the medical and nursing staffs.

Role of the Physiotherapist.—The attentions of the physiotherapist must be devoted to patients who fail, for one reason or another, to maintain adequate exercise. The hopeless task which confronts the physiotherapist of providing adequate supervision of exercises performed by patients confined to bed is thus solved.

General Nursing Care.—Special emphasis should be laid on the care of the bowels. Constipation tends to occur readily from change of diet and environment and causes patients anxiety. Their remedy for this is to practise the Valsalva manœuvre on the bed-pan or in the seclusion of the toilet. This is especially dangerous and is a common cause of detachment of a thrombus from the veins of the lower limbs. Patients should be warned against this dangerous practice and purgatives given at the discretion of the medical and nursing staff. However, there is no evidence that constipation is in itself harmful. The unnecessary use of sedatives must be condemned, as these drugs tend to reduce mobility of the recumbent patient.

Patients confined to Bed prior to Admission.—The risks of venous thrombosis after confinement to bed for more than one week are high and the subsequent treatment of such cases should be planned with this in mind. Sudden changes in the level of basal mobility are dangerous, and this should be the guiding principle. Therefore, graduated soleus muscle exercises should be started, using the weaker spring setting and increasing to the routine level in approximately forty-eight hours. This problem is discussed further when the role of anticoagulant therapy is considered.

ANTICOAGULANT THERAPY

Anticoagulant Therapy in the Prophylaxis of Venous Thrombosis.—

General Principles.—The rationale of anticoagulant therapy in the prophylaxis of pulmonary embolism depends upon the ability of these drugs to prevent or restrict venous thrombosis in the presence of venous stasis.

Prophylactic anticoagulant therapy has been used routinely in surgical cases by Reich, Yahr, and Eggers (1945); Barker, Cromer, Hurn, and Waugh (1945), and Thies (1954). The anticoagulant drugs were found effective in reducing thrombo-embolic complications by all investigators. Reich and others, who began medication on the third and fourth post-operative

day, encountered no post-operative bleeding. However, Thies, who began prophylaxis on admission, encountered a general bleeding frequency of 1 per cent, although this figure was as high as 7 per cent after gastrectomy. No published necropsy data of the efficiency of anticoagulant therapy in the prevention of venous thrombosis are at present available. However, in the limited experience of the author, anticoagulant drugs are very effective, provided they are administered soon after admission to hospital.

Prophylactic anticoagulant therapy in medical cases has been restricted almost entirely to cases of myocardial infarction (Russek and Zohman, 1957; Honey and Truelove, 1957; Toohey, 1958) and congestive heart failure (Anderson and Hull, 1948; Wishart and Chapman, 1948; Harvey and Finch, 1950). All investigators report a reduction in the incidence of thrombo-embolism, and Honey and Truelove state that anticoagulants abolished pulmonary embolism in their series of 543 patients.

The laboratory control of anticoagulant therapy, even with the newer drugs, precludes its routine use at the present time, but it is recognized that some patients present special problems whereby an adequate level of active mobility is difficult or impossible to achieve.

Obesity.—Obese patients are very liable to pulmonary embolism, particularly following abdominal surgical operations (Snell, 1927). The combination of body-weight and the thick investment of adipose tissue in the flexures at the inguinal ligament encourage venous stasis and consequently thrombosis.

Congestive Cardiac Failure.—Prolonged recumbency and œdema of the tissues of the lower limbs promote venous stasis and hence thrombosis. Early ambulation is frequently contra-indicated in cases of myocardial infarction.

Traumatic Injuries and Orthopædic Operations involving the Lower Limb.—This group is most susceptible to venous thrombosis which may begin within two or three days of recumbency. Early active movements may be difficult or impossible. It is most important that necessary surgical intervention should be undertaken as soon as possible after confinement to bed. Manipulation or operation between the second and seventh days is most dangerous and may result in extensive thrombosis in the veins of the lower limb, and pulmonary embolism.

Pregnancy.—Pregnancy exerts severe stress on the veins of the lower limb. Venous thrombosis is particularly liable to occur in obese women with varicose veins during the puerperium, particularly if they are partially immobilized by perineal sutures or following Cæsarean section. Particular attention should be directed to active movement of the lower limbs in bed in these susceptible groups. In addition, it would seem advisable to give routine anticoagulant therapy in an attempt to restrict thrombosis. This treatment should be continued for one week, to allow time for retraction and organization of thrombus already formed. After this time the continuation of anticoagulant therapy should depend on the ability of the patient to perform adequate active movements.

Anticoagulant Therapy in the Treatment of Venous Thrombosis.—The problem concerning the treatment of a venous thrombosis presenting with clinical signs and symptoms is more complex. Barker, Nygaard, Walters, and Priestley (1941) found that 24·3 per cent of the cases of pulmonary embolism showed no symptoms of venous thrombosis, and only 5·2 per cent of fatal emboli were preceded by clinically diagnosed ileofemoral thrombosis.

Pulmonary embolism is often the first sign of venous thrombosis since the thrombus is in the formative stage and only loosely attached to the vein. Sudden increase in the volume of the venous return by compression of the lower limbs during lifting the patient, sudden vigorous movement, or the Valsalva manœuvre may free the thrombus. As soon as obstruction of the lumen of the vein by the thrombus is produced embolism is less likely to occur, but the condition is often not recognized clinically until this stage. Treatment of 'blue' or 'white' legs with anticoagulants is therefore directed to prevent extension of the thrombus into anastomotic veins and thereby lessen the severity of the post-thrombotic syndrome. However, thrombosis is usually bilateral, and where extensive venous occlusion is diagnosed in one limb particular attention should be paid to the apparently normal contralateral lower limb. In the latter, the thrombus may be in the formative stage, and sudden onset of vigorous active movement may detach an embolus from the vein of the 'normal' lower limb.

The clinical approach to this problem is clear. Graded exercises using the weaker spring setting should be started and gradually increased over the next forty-eight hours until the routine basal level is reached. No attempt at ambulation should be made for one week, since organization of the thrombus does not begin until approximately four days after formation. Organization causes the thrombus to adhere to the vein wall so that detachment is unlikely. After one week ambulation should be gradually started and full activity attained in two weeks from the onset. Œdema of the limb may be restricted by firm bandaging during the ambulant phase of treatment.

SUMMARY

A clinical programme is described and discussed for the prophylaxis of pulmonary embolism. This programme is based largely on previous investigations carried out by the author.

The role of the medical, nursing, and physiotherapy staffs is described.

A simple apparatus has been devised for the determination and control of lower limb exercise during bed-rest (the Soleus ergometer).

It is emphasized that the problem of pulmonary embolism will be overcome only by appreciation of the mechanics of venous stasis and the conscientious practice of a controlled prophylactic programme.

Acknowledgements.—I am indebted to Mr. Stanley Cox for his enthusiastic help and for manufacturing the ergometers to my design, and to Mr. R. O. Davies for experimental engineering. I wish to thank Sister S. B. Cooper, who was responsible for the clinical trial of the apparatus, and Mr. H. A. C. Chalmers and Mr. H. G. N. Cooper for their co-operation. The photographs of the ergometer were taken by Dr. L. A. Reitsma and the remainder of the illustrations were produced by kind permission of the

British Journal of Surgery. Finally, my thanks are due to Mrs. E. G. Hudson, who typed the manuscript.

The Soleus ergometer may be obtained from Stanley Cox Ltd., Electrin House, 93-97 New Cavendish Street, London, W.1.

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CHLORPROMAZINE JAUNDICE—A PITFALL IN SURGICAL DIAGNOSIS

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CHLORPROMAZINE hydrochloride (largactil) was introduced into anæsthetic practice in 1951, and it has been used by psychiatrists since 1953. Since then it has been used in ever increasing amounts, but on account of various toxic manifestations a case has been made against the indiscriminate use of the drug (Sherlock, 1958).

Not the least of its undesirable effects is the production of jaundice simulating surgical jaundice, both clinically and biochemically.

Three recent cases brought home the importance of the diagnosis and misdiagnosis of this condition, which is well known to physicians, but to surprisingly few surgeons—there is no reference to the condition in any modern surgical text-book.

CASE REPORTS

Case 1.—Mrs. J. A., aged 80 years, was referred to a surgical clinic complaining of abdominal pain of two years' duration and jaundice from the preceding week. She had had recurrent bouts of sharp pain situated in the right hypochondrium. There was no history of vomiting, but she complained of nausea and a feeling of fullness in the upper abdomen. Her most recent attack had occurred ten days previously. Some three days later friends of hers remarked that she was jaundiced. On direct questioning she admitted that her urine was dark and that she passed pale stools. She also noted itching of the skin.

Her practitioner, in his note, commented that she was "allergic to numerous drugs; penicillin, sulphonamides, barbiturates, etc.".

ON EXAMINATION.—This revealed a well-preserved elderly female who was obviously jaundiced. Apart from several extrasystoles, no abnormal physical signs were detected.

Investigations.—Serum bilirubin, 3·2 mg. per cent; alkaline phosphates, 56 units; thymol turbidity, 0 units; zinc sulphate, 2 units; protein, 7·7 g. per cent—albumin 4·7, globulin 3·0, A : G 1·5; Hb 14·5 g./100 ml.; W.B.C. 9000—differential: neutrophils 77 per cent, lymphocytes 14 per cent, eosinophils 5 per cent, basophils 2 per cent, monocytes 2 per cent.

Cholecystography.—"The gall-bladder failed to outline with contrast medium. No gall-stones seen. After injection of biligrafin, the contrast medium was excreted through the kidneys. Common bile-duct not demonstrated."

During the biligrafin examination the patient developed a marked generalized urticaria and she was given anthisan 100 mg. t.d.s. for 3 days. She was admitted ten days later with the provisional diagnosis of chronic cholecystitis, with stone in the common bile-duct, the diagnosis of obstructive jaundice being supported by the raised serum bilirubin and alkaline phosphatase, as well as normal thymol turbidity and zinc sulphate, and the repeated failure of the gall-bladder to opacify. She was then less jaundiced, and after preparation with vitamin K, laparotomy was performed through a Kocher incision.

AT OPERATION.—The liver was slightly enlarged and had a fine granular surface. The gall-bladder was normal. The common bile-duct was a little dilated, but exploration revealed no abnormality. A T-tube was inserted into the duct, a wedge of liver taken for biopsy, and the wound closed in layers.

The patient's post-operative recovery was quite uneventful, her jaundice lessening each day. On the sixth post-operative day she complained of pain under the right ribs, but no cause could be found. However, because of her persistent pain, and also because of some dysphagia

FIG. 297.—*Case* 1. Showing the well-marked hiatus hernia.

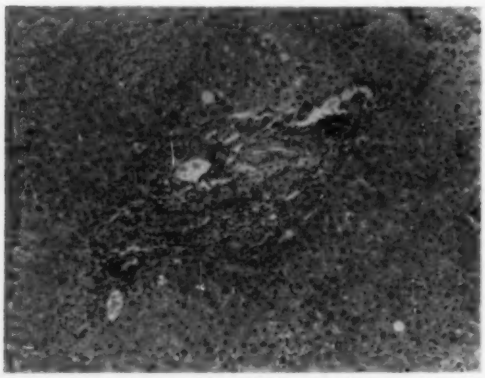

FIG. 298.—*Case* 1. Showing infiltration of the portal tract with inflammatory cells, but with some polymorphs and eosinophils. Note absence of bile-duct proliferation. (× 75.)

for solids, a barium-meal examination was ordered to exclude peptic ulcer or hiatus hernia. The latter diagnosis was quite clearly shown (*Fig.* 297). By this time, examination of the liver biopsy (*Figs.* 298, 299) suggested that the features of toxic hepatitis, which it showed, were similar to those produced by chlorpromazine. The patient's practitioner was consulted and confirmed that she had had 75 mg. t.d.s. for approximately two weeks prior to the onset of jaundice!

The patient responded to conservative treatment for her hiatus hernia and was discharged much improved after three weeks in hospital.

In retrospect, this was a case of hiatus hernia with exacerbations of œsophagitis. Had we been aware of the previous administration of chlopromazine, we might well have avoided an unnecessary laparotomy by ordering a barium-meal examination in the first place.

Case 2.—In March, 1958, E. J. R., a 52-year-old labourer, presented at a surgical clinic complaining of nagging epigastric pain of two months' duration. It was easier at night and was relieved to some extent by alkalis. The pain sometimes radiated round the right flank and into his back. He denied having nausea or vomiting, and complained his weight was steady, although he had a poor appetite.

He was found to be an anxious, well-nourished male. Heart and lungs were clinically clear. In the abdomen there was tenderness to deep pressure over the epigastrium, but no mass was palpable.

A barium meal was arranged to confirm a clinical impression of peptic ulcer, and the patient was ordered an ulcer diet. A fortnight later the barium series showed no abnormality. However, his pain was a little worse and he was "worried stiff".

In view of the radiation of the pain to the right flank, and because he now admitted to some dysuria when directly questioned, an intravenous urogram was arranged. This was negative.

Because the patient was still complaining his practitioner referred him back to hospital. He now claimed that the pain was gripping in nature and that it made him tremble. He no longer obtained relief from his diet and alkalis. Since his first visit he had lost 2 st. in weight.

ON EXAMINATION.—This revealed an anxious, pallid man rolling around with pain. Pulse and temperature were normal. There were no other positive features, and he was admitted for investigation of an anxiety state.

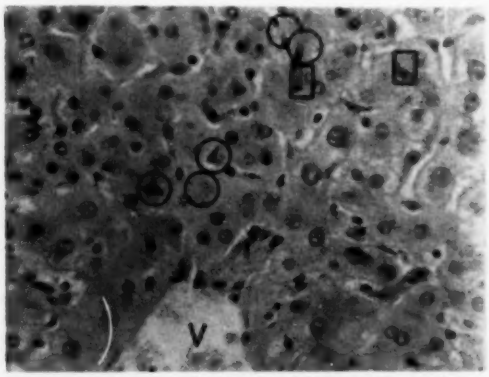

FIG. 299.—*Case* 1. Showing centrilobular area surrounding central vein (V). There is bile-pigment in liver cells, in Kupffer cells (○), and in two bile canaliculi (□). Note also the swelling of the liver cells. (× 300.)

Investigations.—Blood-sedimentation rates, 5 mm., and 16 mm./hr.; Hb 14·1 g. per cent; W.B.C. 7000; urine clear; stools, no occult blood; radiographs: chest and lumbar spine, cholecystogram, repeat barium meal, and repeat intravenous urogram were all negative.

The patient's psychological background was considered to be significant. He was married to a woman ten years his senior and she took full responsibility for the family affairs. He was also having difficulties in his present employment. A psychiatrist arranged his transfer to a lighter job, and he was discharged, taking promazine 50 mg. t.d.s. When seen in the psychiatric department a fortnight later, as all his symptoms had increased, he was admitted to a hospital for nervous diseases for management of his depressive state.

He was given modified insulin shock treatment, without much improvement. Some six weeks after the start of the promazine therapy he became clinically jaundiced. His urine was quite dark and his stools paler than normal.

Investigations at this juncture revealed serum bilirubin, 9·0 mg. per cent; alkaline phosphatase, 51 King-Armstrong units; thymol turbidity, 3 units; zinc sulphate, 4 units.

Liver biopsy showed "bile retention, with polymorphonuclear infiltration of the portal tracts, with relatively severe liver cell damage. There is no bile-duct proliferation." The weight of evidence strongly favoured toxic rather than extrahepatic obstructive jaundice.

However, as there was no improvement of the jaundice after a few weeks, a surgical opinion was again sought. Examination of the patient's abdomen now revealed definite ascites. On the following day laparotomy was undertaken. After the ascitic fluid had been aspirated, the head of the pancreas was found to be the seat of a large inoperable carcinoma, and secondaries were present in the liver. A palliative choledochojejunostomy (Roux-en-Y) was performed.

The patient's condition post-operatively was never very good, and after several ups and downs, despite all efforts to resuscitate him, he died on the fifth post-operative day.

Case 3.—G. H. C., a 66-year-old storekeeper, was admitted on April 23, 1956, for investigation and treatment of an above-knee amputation-stump pain of five weeks' duration, amputation of the left leg having been performed during the 1914-18 war following a gunshot wound. Examination revealed trigger points over the stump and a course of ultrasonic therapy was prescribed. This was followed by some relief and the patient was discharged, to report back in three months.

As his pain had become intractable he was re-admitted on July 26, 1956. In addition to local treatment he had been given 50 mg. largactil t.d.s. since mid-July. In August he was encased in a plaster spica and a week later he complained of feeling unwell. He had passed only a small quantity of dark urine, and had vomited on two occasions.

On Examination.—Temperature was 99·6°, pulse 136, respiration 32. He was definitely jaundiced, and though the plaster spica impeded physical examination, he was found to have an enlarged liver.

Liver function studies at this stage showed serum bilirubin, 3·2 mg./100 ml.; serum alkaline phosphatase, 23 King-Armstrong units; thymol turbidity negative; colloidal gold negative.

The patient was given extra fluids and the largactil was stopped, the jaundice being attributed to its administration. Thereafter the jaundice fluctuated somewhat, but after a few days the patient suddenly had a large hæmatemesis. A surgical opinion was sought, and after a further hæmatemesis laparotomy was considered necessary to stem the bleeding.

The patient's condition, however, deteriorated rapidly and he died two days after the first hæmatemesis on Aug. 29, 1956.

Post-mortem examination revealed that a chronic duodenal ulcer was the cause of the bleeding, and that the jaundice was due to carcinomatous glands in the portal fissure, the source being a clinically silent carcinoma of the lung, which had completely occluded the right lower lobe bronchus and had spread extensively to the subhilar glands.

DIAGNOSIS

In the majority of cases the diagnosis can be made from the history of chlorpromazine therapy, from transient eosinophilia which is sometimes present, and from the liver biopsy, which shows infiltration of the portal tract by inflammatory cells without proliferation of the bile-ducts, together with the presence of bile-pigment in the enlarged liver cells, the Kupffer cells, and in the bile canaliculi (*see Figs.* 298, 299).

However, in doubtful cases and in those in which the jaundice is slow to clear it is wiser to perform exploratory laparotomy. Persistence of jaundice for six weeks has been suggested as a reasonable time interval before embarking on surgery (Read and Sherlock, 1959).

DISCUSSION

Over 800 cases of jaundice appearing both during or soon after chlorpromazine therapy are recorded in the literature. Overt jaundice is said to occur in 1–4 per cent of those exposed to the drug for one week (Doughty, 1955). Its appearance is not related to either the quantity of drug administered or to the length of time for which it is given. There is one case on record in which jaundice appeared after a single dose of 20 mg. (Hollister, 1957). This, together with the eosinophilia and skin rashes which not infrequently accompany or follow the exhibition of chlorpromazine, lends support to the argument that the condition is due to an allergic reaction. (The patient described in *Case* 1 was allergic to several drugs, probably to chlorpromazine as well.)

The other theory of causation suggests that one of the end-products of chlorpromazine metabolism, which occurs in the liver, might be toxic to the biliary radicles (Sherlock, 1958).

The prognosis of chlorpromazine jaundice *per se* is good, but fatalities have been reported. They are usually due to the disease for which the drug has been prescribed. It has been shown that the condition is more serious in pregnant women, many of whom are given chlorpromazine for persistent morning sickness.

CONCLUSIONS

Attention is again drawn to the increasing incidence of chlorpromazine jaundice, which may present clinically and biochemically, indistinguishable from surgical jaundice, and which is still proving a pitfall in surgical diagnosis. McGrath and McNamara (1958) point out that cases will continue to appear in which the diagnosis will be made only after the absence of extrahepatic obstruction is demonstrated by laparotomy.

On the other hand, it must be remembered that jaundice appearing in a patient receiving chlorpromazine is not necessarily due to the drug, but may well be due to surgical obstruction, as illustrated in *Cases* 2 and 3. This is yet another instance when the old aphorism of 'when in doubt, look and see' may usefully be employed.

Acknowledgements.—I wish to thank Professor Ian Aird and Dr. Charles Fletcher of the Departments

of Surgery and Medicine of the Postgraduate Medical School, and Mr. Leon Gillis of Queen Mary's Hospital, Roehampton, for permission to report these cases. My thanks are due to Dr. G. Clennar for drawing my attention to *Case* 3. I owe special acknowledgement to Professor C. V. Harrison of the Department of Pathology, Postgraduate Medical School, for his description of the pathological features, and to Mr. W. Brackenbury of the Department of Medical Illustration for the photomicrographs.

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CONGENITAL SKIN SINUS OF THE PENIS
REPORT OF A CASE

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CONGENITAL anomalies of the male external genitalia are of frequent occurrence, yet the minor ones involving the perineal raphe appear to be rather rare, and search of the literature reveals that less than 50 cases of congenital cysts and canals have been described since the beginning of the century. Possibly the cysts occurring in this region have been overlooked, or mistaken for sebaceous cysts or the like, but congenital sinuses of the perineal raphe are certainly rare, and the case described reveals how such sinuses usually come to light, namely, when infection supervenes. It is more than likely that these canals are commoner than the literature indicates, but are never noticed throughout life simply because they remain quiescent.

CASE REPORT

J. H. A., a healthy seaman aged 22 years, reported to a venereal disease clinic in July, 1958, with an intermittent discharge from the underside of the shaft of the penis. He stated that following intercourse one year previously a swelling appeared on the ventral surface of the penis which burst and discharged pus, and about three weeks later another swelling appeared more distally, and this also burst and discharged pus. Since then there had been an intermittent discharge from both sites of the original swellings. Apart from this complaint he was fit and had no other symptoms, there being no disorder of micturition.

Examination revealed that he was uncircumcised, and on the ventral surface of the penis were two minute orifices in the midline, one about 1 in. from the prepuce, and the other about 2½ in. more proximally. It was possible to pass a fine lachrymal probe into one orifice, along the sinus track, and out at the other orifice (*Fig.* 300). Massage of the track expressed some light yellow pus, a Gram film showing scanty Gram-positive cocci, culture of a swab revealing coagulase-negative staphylococci and a few diphtheroids. No gonococci were demonstrated and dark-ground examination was negative. Wassermann and Kahn reactions were also negative.

It was decided that this was a case of non-venereal infection of a congenital sinus, and the patient was therefore referred for a surgical opinion, when excision was considered to be the correct treatment. At operation the sinus was excised, after first passing a probe, and the skin closed. The patient made an uneventful recovery with perfect healing of the skin.

The histological report on the excised specimen reads as follows: "Sections show a sinus track lined by stratified squamous epithelium, closely resembling the normal epidermis, save in the absence of skin appendages. The

A B

FIG. 300.—A, The ventral median skin sinus, visible as a linear marking which is slightly less pigmented than the surrounding penile skin; B, With a probe in situ.

appearances are consistent with the diagnosis of congenital midline skin sinus."

DISCUSSION

Congenital anomalies of the perineal raphe are of considerable interest to the embryologist as the development of the external genitalia in the male is still not entirely clear. The perineal raphe, which is prominent on the scrotum but less so on the ventral surface of the penis, is a permanent surface-marking, indicating where fusion of the primitive urethral crests took place during embryonic development, the enclosed canal forming the urethra. The mucosa of this canal is formed from endoderm which lies deep in the urethral groove, and which is continuous with the mucosa of the urogenital sinus. It follows, therefore, that in this process of fusion of the urethral crests, some epithelial cells might be enclosed in the plane of

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fusion, analogous to the formation of branchial cleft cysts, and leading to the formation of canals or cysts. The urethral crests, however, are covered by ectoderm on their outermost part, which explains why a superficial canal (such as the one described) is lined by squamous epithelium. The most terminal part of the urethra, that which lies in the glans penis, is formed by the urethral plate, a mass of ectodermal cells which grow inwards and meet the endodermal canal already formed. This later becomes canalized, and it is clear that abnormalities arising from this area, such as cysts and canals, would be lined by squamous epithelium.

This is the usual explanation of the origin of abnormalities of the perineal raphe, but Neff (1936) puts forward another theory, which assumes migration of either urethral endoderm or surface ectoderm along the line of fusion, leading to the formation of either cysts or canals. In support of this theory one investigator showed that in an 88-mm. embryo there were some cellular outgrowths from the penile urethra completely split off, and some in the process of being divided from the main epithelium, both types being already canalized. This would explain, he states, the formation of peri-urethral cysts and ducts.

In a recent article Glenister (1958) describes the formation of the penile urethra in great detail, and explains how the primary urethral groove is deepened by a secondary urethral groove, the two together being known as the 'definitive urethral groove'. The whole groove is flanked by the definitive urethral folds, and the junction of the two types of epithelium is clear-cut. This modification of the earlier view on the embryology of this region assists in the understanding of how midline abnormalities can arise.

As already mentioned, cysts and especially canals of the perineal raphe have received scant attention in the literature. Balzer and Souplet in 1893 were apparently the first to describe 2 cases of midline sinuses, but two years later Mermet (1895) had collected 23 cases of cysts and investigated them histologically. He found that 16 had a lining of squamous epithelium, and 4 a lining of cells of endodermal origin. The remaining 3 were unspecified. Rupel (1924) reported 8 cases of epidermal canals infected with gonococci, and in 1929 Okawa reported a further 9 cases, stratified epithelium being found in them all. Gougerot, Burnier, and Tissot in 1933 described 11 cases of midline canals, stratified squamous epithelium lining all but 2, and Neff, in 1936, brought the total number of reported cases of congenital cysts and canals of the perineal raphe to 33. Both Neff and Lamb, who reviewed the subject in 1943, stress the frequency of mistaken diagnosis, stating that when the canals are infected the lesions are easily confused with chancres, chancroidal infections, tuberculous sinuses, pyogenic infections, lymphogranuloma venereum, and the like. These authors emphasize the fact that these cases often arrive in the first instance in a venereal clinic (as in the present case). In one of Neff's cases the original lesion appeared at the frænum of the prepuce. Wooldridge (1955) reports a further 3 cases of canals, and states that these canals are exclusively lined by stratified squamous epithelium, and mentions that very rarely a pilonidal type of sinus may exist, containing hair. A case has been reported of a congenital sinus of the prepuce which was lined by squamous epithelium at its origin and further down the duct by a columnar cell structure.

Cysts of the perineal raphe are probably more common than sinuses, but are not reported owing to errors in diagnosis and frequency of confusion with sebaceous cysts. They are classified as either mucous cysts, in which case they are lined by columnar epithelium, or dermoid cysts, in which case the lining is squamous. These cysts may be located anywhere along the raphe from the prepuce to the anus. One investigator examined 500 male infants and in 3 cases only found a small cyst with an epidermoid lining near the frænum of the prepuce.

SUMMARY

A case of midline sinus of the perineal raphe is described and the embryology is discussed. A review of the literature reveals the comparative rarity of the condition.

Acknowledgements.—I am indebted to Mr. J. P. Hosford and Dr. C. S. Nicol for permission to publish this case, and to Mr. E. G. Tuckwell for helpful advice and criticism.

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THE RUPTURED ABDOMINAL AORTIC ANEURYSM AND ITS TREATMENT

By G. E. MAVOR, L. D. DAVIDSON, and C. G. CLARK

ABERDEEN ROYAL INFIRMARY

Elective surgery in the presence of an abdominal aortic aneurysm which is not producing symptoms is now accepted as justifiable, and the best treatment is resection of the aneurysm and reconstitution of the aorta by a homograft or plastic prosthesis (Bahnson, 1954; De Bakey and Cooley, 1953; Rob, Eastcott, and Owen, 1956). The problem of treatment becomes more complicated when the patient is first seen with the aneurysm ruptured. In most instances there is an interval between the initial retroperitoneal hæmorrhage and the final fatal collapse. This interval may often be of several days' or even weeks' duration. Usually it is not less than twelve hours (Barratt-Boyes, 1957). The opportunity thus presented to carry out resection of the aneurysm and restoration of the aortic channel must be taken, as the patient's best chance of survival lies in the success of such surgery.

Four cases of ruptured aneurysm of the abdominal aorta were seen over the past three years, and in each reconstructive aortic surgery was undertaken. One patient survived three months, while another is alive and well one and a half years after surgery.

CASE REPORTS

Case 1.—G. A., aged 58 years, was admitted to hospital complaining of lumbar pain and vomiting. These symptoms began ten days previously. The pain was of sudden onset, severe and aching in nature, and situated in the left lumbar region. It did not radiate. The patient went to bed and remained there. Two days later he vomited small amounts of greenish material, and this vomiting continued several times a day for three or four days. After the first day the pain subsided, but did not disappear. Over the last few days he had noted vague abdominal discomfort. There were no other recent symptoms of significance.

He had been in hospital on three occasions during the past six years with coronary heart disease and essential hypertension. Two years previously, when last seen, he had had severe angina of effort, being able to walk only 50 yards at a slow speed without chest pain. His heart was enlarged, and electrocardiography showed an old posterior myocardial infarction. Blood-pressure then was 240/130 mm. of mercury. There was no abnormality noted on abdominal examination.

On Examination.—The patient was very distressed and in severe pain. There was marked cardiac enlargement with a generalized systolic murmur. The blood-pressure was 240/100 mm. of mercury, and the pulse-rate 70 beats per minute. The left femoral pulse was noted to be weaker than the right. In the abdomen there was a large, firm, smooth, pulsatile mass extending from the left costal margin to the left lower abdomen. The diagnosis of an abdominal aortic aneurysm was made.

Urinalysis showed no abnormality apart from a trace of albumin; stool benzidine was negative. Hæmoglobin was 102 per cent, erythrocyte sedimentation rate 12 mm. in the first hour, Wassermann reaction negative, and blood-urea estimation was 38 mg. per 100 ml. The electrocardiogram showed left ventricular strain and myocardial ischæmia; radiography of the chest revealed an aneurysm of the descending aorta, and of the abdomen, a left-sided soft-tissue shadow suggesting an abdominal aortic aneurysm; a barium-meal examination demonstrated that the aneurysm displaced the third and fourth parts of the duodenum forwards and to the right.

Five days after admission to hospital the patient was seen in surgical consultation. During this period his condition had not varied. Slight abdominal discomfort was present, but there was no lumbar pain and his blood-pressure was maintained. Operation was advised and planned for three days later, the delay being due to the need to arrange facilities for general hypothermia. However, during the evening of the second day the patient developed severe back pain associated with vomiting. He complained of very cold feet and this was rapidly followed by marked collapse with sweating and peripheral cyanosis. His blood-pressure was 120/90 mm. of mercury. Blood transfusion was started, and the patient received 4 pints of whole blood during the three hours prior to operation.

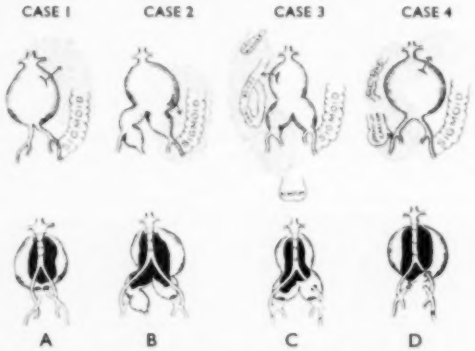

Fig. 301.—Diagrammatic representation of aneurysm, site of rupture, and extravastion area in each case. The aortic reconstitution is also shown.

At Operation.—Operation was started 17 days after the onset of symptoms, 7 days after admission to hospital, and 4 hours after the onset of severe collapse. At the onset the blood-pressure was 90/60 mm. of mercury. Anæsthesia was induced with thiopentone 150 mg. and succinylcholine 50 mg., and maintained with nitrous oxide, oxygen, and ether via an endotracheal tube. General hypothermia was induced by surface cooling. During the operation the lowest rectal temperature recorded was 29° C., while the pharyngeal temperature did not fall below 29·5° C.

On opening the peritoneal cavity a large retroperitoneal hæmatoma was noted mainly on the left side, although there was also marked extravasation in the upper abdomen. About two pints of blood-stained fluid were present in the peritoneal cavity. The aneurysm involved the origins of both renal arteries, but inferiorly it did not extend beyond the aortic bifurcation, both common iliacs being of normal calibre (*Fig.* 301 A). The latter were mobilized and controlled. Dissection at the upper aspect of the aneurysm was made difficult by the extensive extravasation, and considerable bleeding occurred before the aorta was finally controlled and clamped above the renal arteries. Thereafter the common iliac arteries were clamped, and the aneurysm incised through its anterior wall. The anterior wall of the aneurysm was excised and old blood-clot in the aneurysm evacuated. The lateral and posterior walls of the aneurysm were left in situ. The posterior wall of the aneurysm was divided across below the renal arteries enabling proximal dissection of the posterior wall. In this way a cuff of aorta was prepared below the renal arteries.

At this stage the aortic clamp was re-applied below the renal arteries. An arterial homograft was used for the aortic reconstruction. The proximal anastomosis entailed considerable 'tailoring' of the aorta. The right lower anastomosis was then fashioned end-to-side to the right common iliac artery, and the blood-flow to the right leg restored by removal of aortic and right common iliac clamps, the left limb of the homograft being clamped. The duration of aortic occlusion was 1 hour and 50 minutes. Up to this time the patient received 6 pints of blood, making a total of 10 pints transfused. The systolic blood-pressure varied between 85 and 100 mm. of mercury. During the suturing of the left lower anastomosis, also end-to-side to the left common iliac artery, cardiac arrest occurred. Resuscitation was not successful. The duration of the operative procedure was 3½ hours.

Case 2.—W. F., aged 65 years, was admitted to hospital complaining of severe abdominal pain and collapse. Thirty-six hours previously he had experienced discomfort in the left buttock and thigh. Although this had persisted, it was not of great severity, nor did it become worse. There was no vomiting or other complaint. Three hours before admission, he developed severe abdominal pain while at supper, and shortly afterwards collapsed. He had passed no urine over the previous eleven hours.

On Examination.—The patient was severely shocked, cyanotic, and cold. Temperature was 95° F., and pulse-rate 110. The pulse was very weak. The blood-pressure was 50/0 mm. of mercury. Heart sounds were faint. A large pulsating abdominal mass was present, more prominent to the left, and reaching well above the umbilicus. Both femoral pulses were present and equal. The diagnosis of a ruptured aortic aneurysm was made. Blood transfusion was begun and arrangements completed for laparotomy. A urinary catheter was passed, but less than 10 ml. of urine obtained.

At Operation.—Laparotomy was started 38 hours after the onset of symptoms, 5 hours after the onset of collapse, and less than 2 hours after the patient's admission to hospital. At the onset the blood-pressure was 50/30 mm. of mercury, the patient having received 3 pints of blood during the pre-operative period. Anæsthesia was induced with thiopentone 150 mg. and succinylcholine chloride 50 mg., and maintained with nitrous oxide and oxygen via an endotracheal tube and 50 mg. *d*-tubocurarine chloride given over 7 hours.

The abdomen was opened through a long left para-central incision. A large abdominal aortic aneurysm was revealed with extensive extravasation of blood to the left involving and covering the descending and sigmoid colons. The pelvic mesocolon was 3-4 in. thick. The aneurysm extended to within an inch of the renal arteries, and inferiorly both the common iliac arteries were aneurysmal as was the right internal iliac artery (*Fig.* 301 B). Through a separate chest incision the thoracic aorta was mobilized and clamped. After division of the parietal peritoneum over the aneurysm and reflection of the duodenum to the right, the aorta below the renal vessels was mobilized by finger dissection, and a clamp applied. The clamp on the thoracic aorta was now removed. It had been in place for about 30 minutes. Both external iliac arteries were now mobilized and clamped, as was the left internal iliac artery. At this stage the aneurysmal right internal iliac could not be safely mobilized. The aortic aneurysm was incised and evacuated. Only a small trickle of blood escaped from the right internal iliac artery aneurysm, and this was controlled by packing. A good aortic cuff was then prepared below the renal arteries and the proximal anastomosis carried out. Again, 'tailoring' of the aortic homograft was necessary. Distally, the right anastomosis—end-to-side homograft to external iliac artery—was completed first, the external iliac artery being ligated proximal to the anastomosis. The blood-flow to the right leg was now restored. The period of aortic occlusion below the renal arteries was three hours. The right internal iliac aneurysm was now incised and with a finger in it, dissection was carried down into the pelvis, where the vessel became of normal calibre. It was ligated. The distal anastomosis on the left side was now completed, again end-to-side homograft to external iliac artery. Restoration of flow to the internal iliac artery was possible (*Fig.* 301 B). The aortic aneurysm was left in situ except for the anterior wall, which was excised (*Fig.* 301 B). Heparin was used locally in all anastomoses and injected distally into the lower limb arteries. The arrest of blood-flow to the left leg lasted four hours. Before closure of the abdomen the sigmoid colon was observed to be of good colour. It was noted during the operation that there was abnormal resistance to inflation of the lungs, with diminution of elastic recoil. It was not appreciated at the time that these findings were due to emphysema.

During the course of the operation, which lasted six and a half hours, the patient received a further 8 pints of blood. At the completion, blood-pressure was 100/80 mm. of mercury, pulse-rate 130, hæmoglobin 86 per cent, and urinary bladder content 60 ml., of specific gravity 1014.

Post-operative Progress.—At the end of the operation respirations were not satisfactory, despite the administration of atropine 1·3 mg. and neostigmine 5 mg. The patient's colour was good and he responded to questioning. Despite the administration of oxygen, the patient's condition had deteriorated markedly by the time he reached the ward. He was cyanosed and the blood-pressure was 80/60 mm. of mercury. He was reintubated and his lungs inflated with oxygen with marked improvement in his condition and return to consciousness. It was not until five hours after operation that his respirations and blood-pressure became stabilized. At this point it was considered that his respiratory difficulties were due to persistent curarization caused by failure of the kidneys, or to so-called neostigmine-resistant curarization, or to excessive accumulation of plasma carbon dioxide.

Over the next twelve hours the patient's condition remained satisfactory. The blood-pressure averaged 110/74 mm. of mercury, and the pulse-rate fell progressively from 110 to 94. Hæmoglobin was 96 per cent. Urinary output totalled 80 ml., the specific gravity being 1014. He was conscious and co-operative, and could move his legs and toes. All the peripheral pulses were easily palpated and there was no evident ischæmia of the lower limbs. A radiograph of the chest showed some patchy densities in the lower part of the left lung fields, but no gross abnormality was noted.

During the next 12-hour period (which was overnight) the patient's condition deteriorated rapidly. He became restless and cyanosed, the respiratory rate increasing to over 40 per minute. Despite active respiration with oxygen his respiratory distress continued. The blood-pressure fell gradually throughout the night, failing to respond to noradrenaline and further blood transfusions. He died 30 hours post-operatively.

Post-mortem Examination.—The significant findings were related to the thoracic and abdominal cavities. The pleural cavities contained no free fluid. Both lungs showed a very gross degree of emphysema and in addition massive collapse was present involving both lower lobes. Some focal collapse was also noted in the middle and upper lobes and tenacious mucus could be expressed from many of the cut ends of the bronchi. Examination of the abdomen showed a striking greenish discoloration of the sigmoid colon indicating well advanced gangrenous changes. The kidneys were normal in size and showed moderate nephrosclerotic changes on section. The aortic homograft and all its suture lines were intact.

Case 3.—G. McP., aged 63 years, was admitted to hospital complaining of right-sided abdominal pain present for eight hours. During the night he experienced severe sudden pain in the right iliac fossa. Later, radiation of the pain occurred to the back, right groin, and anterior

thigh. There was no vomiting. As time passed the pain became less severe, but he noted that his right foot was cold and numb. Two hours before admission the pain increased in severity and he felt cold and weak. He passed no urine in the eight hours prior to admission.

On Examination.—The patient was cyanosed and in peripheral vascular failure. Temperature was 97·8 F., pulse of good volume and rate 76, blood-pressure 60/50 mm. of mercury. Heart sounds were faint. A large pulsating abdominal mass was present, much more marked to the right, where there was tenderness and resistance to palpation. Both femoral pulses were present, the right being much weaker than the left.

The diagnosis of a ruptured abdominal aortic aneurysm was made. Blood transfusion was started and arrangements for surgery completed. A catheter was passed and 40 ml. of urine removed. This showed no blood, but a trace of albumin.

At Operation.—Laparotomy was started ten hours after the onset of symptoms and less than two hours after the patient's admission to hospital. At the outset blood-pressure was 70/60 mm. of mercury, the patient having received 800 ml. of blood during the pre-operative period. The patient was only semiconscious when he reached the anæsthetic room and since this was most likely due to cerebral hypoxia associated with hypotension the use of thiopentone was avoided. After 3 minutes pre-oxygenation, anæsthesia was induced by nitrous oxide 8 litres per min. and oxygen 2 litres per min., followed by *d*-tubocurarine chloride 30 mg. 2 minutes later. When apnœa occurred an endotracheal tube was passed and anæsthesia was maintained with nitrous oxide 2 litres per min. and oxygen 1 litre per min., with a Waters' CO_2 absorber and *d*-tubocurarine chloride to a total of 50 mg. Apnœa and controlled manual ventilation were continued for the duration of the operation. Muscular tone and respiratory movements were completely restored with atropine sulphate 1·3 mg. and neostigmine 5 mg. (The patient was conscious and speaking before leaving the theatre.)

The abdomen was opened through a long right para-central incision. There was a massive retroperitoneal hæmatoma with extensive extravasation of blood to the right into the flank and pelvis, and small-bowel and gall-bladder mesenteries. About 2 pints of blood were present in the peritoneal cavity. The aneurysm reached to within 1 in. of the renal arteries. Both common iliac arteries were aneurysmal and both internal iliacs enlarged. The external iliac arteries were healthy (*Fig.* 301 C). After division of the parietal peritoneum over the aneurysm, the aorta below the renal vessels was mobilized by finger dissection, a finger being passed round the aorta without difficulty. Thereafter a Crafoord clamp was applied just below the renal arteries. The external and common iliac arteries were then mobilized and clamped. The aneurysm was incised and the clot evacuated. A small area of the anterior wall of the aneurysm was removed. A cuff of aorta was prepared below the renal arteries and the proximal anastomosis carried out. The right distal anastomosis was now fashioned to the proximal external iliac artery (*Fig.* 301 C), a clamp being placed on the artery proximal to the site of the anastomosis. The external iliac artery was opened just above the inguinal ligament and the vessel washed out with heparin in saline solution. A consider-able amount of clot was removed from the artery proximal to the arteriotomy. The arteriotomy wound was closed, and after removal of the appropriate clamps blood-flow restored to the right leg. The period of aortic occlusion was 2½ hours. The right common iliac artery was now mobilized, sectioned just proximal to the bifurcation, and its distal end closed (*Fig.* 301 C). The left lower anasto-mosis was now constructed at the distal end of the common iliac artery (*Fig.* 301 C). Again, via a separate incision just proximal to the inguinal ligament, some clot was removed from the external iliac artery. For con-venience, the left limb of the homograft was brought down inside the left common iliac artery aneurysm (*Fig.* 301 C). The period of cessation of blood-flow to the left leg was 3½ hours. Large pieces of clot in the aortic and common iliac aneurysms were left in situ, as was its grossly atheromatous and calcified wall. In all anasto-moses heparin in saline solution was used, and 20 ml. was injected distally into the lower limb arteries.

The operation lasted 5½ hours. The patient received a total of 8 pints of blood (*Fig.* 302); 10 ml. of calcium gluconate were also given post-operatively. At the completion of the operation blood-pressure was 180/110 mm. of mercury, pulse-rate 108, hæmoglobin 95 per cent, and urinary bladder content 40 ml.

Post-operative Progress.—To forestall the develop-ment of an ileus, the patient received nothing by mouth

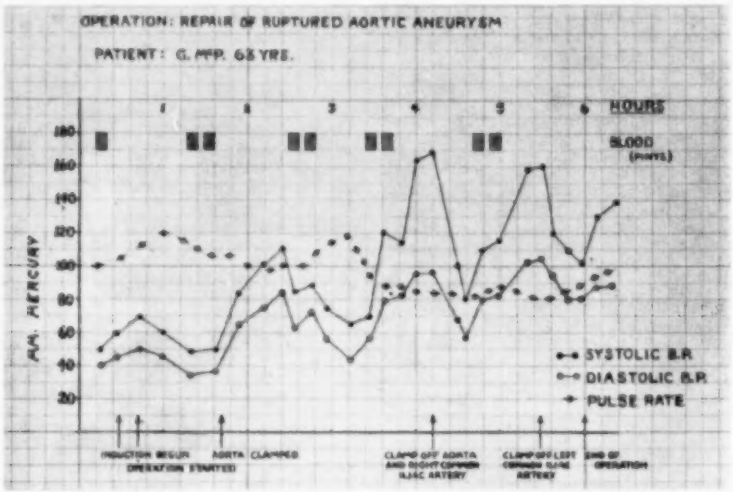

Fig. 302.—*Case* 3. Pulse and blood-pressure chart during the course of operative repair of ruptured aortic aneurysm.

for 5 days. During this period his fluid and electrolyte requirements were met by intravenous therapy. It was not until the morning of the fourth post-operative day that both feet appeared normal, and peripheral ankle pulses were palpable. Prior to this, pallor, coldness, some hypæsthesia, and definite weakness were present, more marked initially on the right foot, but latterly on the left, which took longer to recover. Some impairment of renal function was also present (*Fig.* 303). Otherwise, convalescence was uneventful, and the patient was discharged on the twenty-ninth post-operative day. Seen ten months after operation there was evidence of some impairment in renal function shown by a slight rise in blood-urea (68 mg. per cent) and impairment in maximum concentrating power. This was considered most likely due to renal atherosclerosis. One and a half years after surgery the patient was well with normal arterial pulsations in the lower limbs. He had returned to his work as a slater.

Case 4.—W. A., aged 68 years, was admitted to hospital because of left-sided backache and abdominal pain. This had been present for five weeks. There was no history of sudden onset. The back pain was severe at times and did not radiate. However, it remained persistent and became progressively worse. It was always more severe than the abdominal discomfort. Neither pain was relieved by analgesics or heat. There was no vomiting.

The patient had been in hospital on several occasions, but most recently four weeks previously. At that time he was suffering from backache which had been present intermittently from a back injury sustained fourteen years previously. Over the previous four days it had become more severe and more persistent, situated more to the left than previously. There were no other symptoms of significance. Physical examination showed an ill-looking barrel-chested man suffering from chronic bronchitis and emphysema and limitation of movements of the lumbar spine. No other abnormality was noted and, in particular, no abdominal masses were palpated.

Several investigations were undertaken. Radiological examination of the chest, dorsolumbar spine, and pelvis and intravenous pyelography were negative. Full blood-picture and marrow smear showed no abnormality. The serum urea, serum protein, acid, and alkaline phosphatase were normal. The urine showed a mild coliform infection which was successfully treated by sulphonamides. The only abnormality noted in the course of investigation was a persistently high erythrocyte sedimentation rate which varied between 80–100 mm. per hour. In spite of this, no organic lesion could be found and the patient was discharged to be seen as an out-patient in four weeks' time.

In due course he was seen as an out-patient. He had suffered continually from severe backache. Examination revealed a large pulsatile abdominal mass to the left of the midline. A diagnosis of abdominal aortic aneurysm was made and five days later he was re-admitted to hospital. On admission, examination showed that he was in considerable distress because of back pain. The blood-pressure was 178/104 mm. of mercury and the pulse-rate 64 per minute. The heart was not enlarged. A large abdominal aortic aneurysm was present.

The patient continued to have left-sided backache and this suddenly became more severe during his second hospital day. He was in great discomfort from that time. During the evening of his third hospital day he suffered from epigastric pain radiating to the back, and shortly afterwards he collapsed. The blood-pressure was 80/60 mm. of mercury. There was marked peripheral cyanosis and circulatory collapse. The abdomen was essentially soft. During the night his blood-pressure rose and the peripheral circulation improved to some extent. Twelve hours after the initial episode of peripheral circulatory collapse, the blood-pressure again fell. Shortly afterwards the patient was seen in surgical consultation. At that time, though he was still conscious, marked peripheral circulatory failure was present. No pulse was palpable and the blood-pressure was unrecordable. The abdomen was distended and tender. No pulsating mass was noted. He had passed 100 ml. of urine over the previous twenty-four hours. Immediate arrangements were made for surgical treatment, the pre-operative diagnosis being a ruptured abdominal aortic aneurysm.

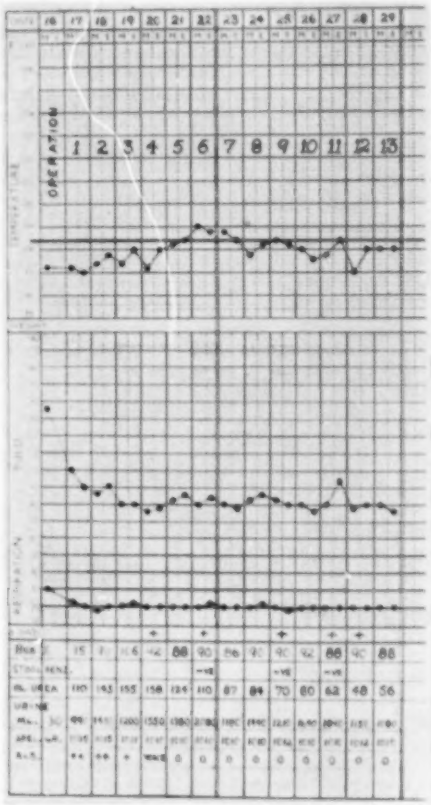

FIG. 303.—*Case* 3. Post-operative temperature, pulse, and respiration chart. Details are also given of hæmoglobin level, stool benzidine tests, serum urea in mg. per cent, and urine.

AT OPERATION.—Laparotomy was started 4 days after admission to hospital, 2½ days after the onset of symptoms of rupture, and 14 hours after the onset of severe collapse. At the outset the blood-pressure was 50/0 mm. of mercury and the pulse-rate was 62 beats per minute. Rapid operative intervention was required and no time was wasted in pre-operative blood replacement.

Anæsthesia was achieved (as in *Case* 3) by nitrous oxide 80 per cent and oxygen 20 per cent following 4 minutes' pre-oxygenation with 100 per cent oxygen, being supplemented by 50 mg. of thiopentone as the patient had a well-marked stage of excitement; 30 mg. of *d*-tubocurarine chloride were given and intubation carried out. No more thiopentone was given during the operation. Anæsthesia was maintained with nitrous oxide 2 litres and oxygen 1 litre per minute with a Waters' CO_2 absorber; 85 mg. of *d*-tubocurarine were given over 7 hours. Apnœa and controlled respiration were continued for the

whole of the operation. No difficulty was encountered in restoring muscular tone and adequate respiratory exchange with atropine sulphate 1·2 mg. and neostigmine 5 mg. A fair quantity of mucopurulent secretion was aspirated from the tracheobronchial tree at the end of the operation. Tracheostomy was considered, but not carried out. The patient was fully conscious before leaving the theatre.

The abdomen was opened through a long left paracentral incision. There was extensive retroperitoneal extravasation of blood involving the superior aspect of the aneurysm, the transverse mesocolon, the mesentery, and the pelvis. The sigmoid colon was not involved. About 3 pints of blood were estimated to be in the peritoneal cavity. The aneurysm did not reach the renal arteries. Inferiorly it involved the aortic bifurcation and the left common iliac artery was fusiform and markedly atheromatous (*Fig.* 301 D).

FIG. 304.—*Case* 4. Retroperitoneal collection of sterile fluid and debris surrounding aortic homograft.

Mobilization of the proximal aorta was carried out by finger dissection, and control of the aorta below the renal arteries and of the external iliac arteries was established. Forty ml. of heparin in saline solution were injected distally into each peripheral arterial tree. The aneurysm was incised and most of the clot evacuated. The proximal aortic cuff was then fashioned, two lumbar arteries being divided in the process. Reconstitution was carried out using an arterial homograft (*Fig.* 301 D).

The period of aortic and right common iliac artery occlusion was 3½ hours, and that of left common iliac occlusion 4 hours. The operation lasted 6½ hours.

After induction of anæsthesia two intravenous infusions were set up (one in each arm), 3 pints of blood were given in the first hour of operation before aortic occlusion, and a further 4 pints were given in the remaining 5½ hours, making a total of 7 pints; 10 ml. of calcium gluconate were given post-operatively. At the completion of the operation the blood-pressure was 120/78 mm. of mercury, pulse-rate 100, hæmoglobin 64 per cent, and urinary bladder content 40 ml. The specific gravity of the urine was 1024 and it contained albumin.

POST-OPERATIVE PROGRESS.—The patient's post-operative course was marked by various complications. Most important among these was the early post-operative development of an acute tracheobronchitis associated with a large amount of mucopurulent sputum. Despite early tracheostomy (carried out on the second post-operative day), antibiotic therapy, and careful physiotherapy, the development of severe bronchopneumonia was not prevented, and for 7 weeks the patient's condition was very grave, owing to persistent pulmonary infection.

FIG. 305.—*Case* 4. Post-mortem specimen showing homograft with remains of posterior and lateral walls of aneurysm. The suture lines are intact and the homograft is unaltered from the time of insertion eleven weeks previously.

Further complications were rupture of the abdominal wound in its upper part (resutured under local anæsthesia), deep venous thrombosis affecting the right leg (treated by anticoagulants over a period of 7 days), and cardiac failure.

To forestall the development of an ileus, nothing was given by mouth for the first five post-operative days, fluid and electrolyte requirements being met by intravenous therapy. After a further 2 pints of blood were given, the hæmoglobin was maintained at 80 per cent. Later, during the course of his illness he received 3 pints of blood. Recovery of renal function was uneventful, diuresis occurring in the third post-operative day, when the urinary output was 1700 ml. (specific gravity 1018 with a trace of albumin). The serum urea was 112 mg. per cent on the second post-operative day, but thereafter fell, reaching a normal range by the end of the first week. Recovery of the peripheral circulation in the lower limbs was without incident, although the posterior tibial pulses were not definitely palpated until the fourth post-operative day.

The patient was nursed through the various post-operative complications and appeared much improved during the course of the eleventh post-operative week, when eventual recovery was expected. However, one afternoon he suffered sudden loss of consciousness and thereafter respiratory distress. Clinically, it was considered that his symptoms were due to a cerebral vascular accident, although they were by no means typical. He failed to regain consciousness and died two days later.

POST-MORTEM EXAMINATION.—Transection of the cerebral hemisphere revealed a 4 × 3 c.mm. hæmorrhage just deep to the left insular cortex, destroying the external capsule and claustrum and involving the lateral part of the putamen. Around the area of hæmorrhage there was cerebral œdema and the vessels showed notable arteriosclerotic changes. The lungs showed evidence of old bronchopneumonia and a mild tracheobronchitis was present. The tracheostomy wound was not completely healed. In the abdomen the small intestine showed occasional scattered small ulcerated areas surrounded by erythema. Their significance was not understood. The sigmoid colon showed a diverticulosis, but apart from this was normal. The adrenals showed evidence of cortical hypertrophy. The kidneys appeared normal. A large fluctuant swelling was found in the retroperitoneal tissue posterior to the root of the mesentery. It was filled with yellowish, soup-like fluid and its walls were lined with shaggy brownish material, presumably altered blood-clot (*Fig.* 304). Foci of calcification were present in the wall of the cavity which extended from the upper aortic suture line superiorly, to the brim of the pelvis inferiorly, with extensions around the first part of the iliac vessels on both sides. Lying within this cavity posteriorly was the aortic graft, patent and with intact suture lines (*Fig.* 305). Part of the wall of the cyst surrounding the aortic graft was formed by the wall of the aneurysm and the rest by thickened retroperitoneal connective tissue. The contents consisted largely of altered blood, but much of the fluid appeared to be purulent. Microscopic examination showed mainly cell debris and "a very few pus cells". Bacteriologically, only a coliform growth was produced, almost certainly due to post-mortem contamination. At the suture lines fibrosis had occurred. At the site of the anastomosis in the subjacent aortic wall a notable foreign body reaction to the suture material was present. The proximal aorta was atheromatous. Macroscopically and microscopically the homograft showed no change in structure from the time of its insertion eleven weeks previously.

ANÆSTHETIC MANAGEMENT

Patients with ruptured aortic aneurysms presenting for aortic reconstruction are rarely in good physical condition. *Case* 1 had two previous episodes of coronary thrombosis. *Cases* 2 and 4 had gross pulmonary emphysema and fibrosis and the first three patients were overweight. With the development of peripheral vascular failure due to massive hæmorrhage the overall picture is not encouraging.

Anæsthetic Agent.—The prolonged nature of the operation and the need for absolute abdominal relaxation necessitate the use of a non-toxic anæsthetic agent and a muscle relaxant. Such a combination is described by Gray and Riding (1957) for anæsthetizing patients for mitral valvotomy. The method was used in *Cases* 3 and 4 and proved satisfactory.

It has been shown beyond question that nitrous oxide is non-toxic when there is no concomitant oxygen lack. The pre-oxygenation used in this method is of immense value to the patient. In the shocked patient, induction is achieved with ease with not less than 20 per cent oxygen in the inspired gases, and the amounts of oxygen can soon be increased to 50 per cent. Complete relaxation and apnœa have been attained by the use of *d*-tubocurarine chloride. The apnœa, by obviating the need for respiratory effort, eases the strain by respiration on the patient and may be an important factor in such gravely ill patients.

The use of *d*-tubocurarine chloride raises the question of renal elimination of this drug in the hypotensive patient, as the drug appears to be excreted mainly by the kidneys, though part is detoxicated in the liver. There is, too, evidence to show that detoxication or inactivation of the drug may occur to a considerable extent in other tissues, particularly in skeletal muscle (Harroun, Beckert, and Fisher, 1947; Wall, 1947). In *Cases* 3 and 4 there was no difficulty in restoring voluntary muscle function, and in retrospect it was considered that the difficulty in restoring respiratory function in *Case* 2 was not associated with the use of the drug.

Hypothermia.—Because it is not possible to investigate the patient pre-operatively, the situation of the aortic lesion in relation to the renal vessels cannot be determined. In such circumstances it is ideal to induce hypothermia in such a way that cooling can be discontinued, should laparotomy reveal that the renal arteries are not involved. This can only be achieved by the water blanket method, the patient lying on the blanket. The operation can then be started at the same time as cooling. Delay is avoided and cooling will have reached a satisfactory level by the time suprarenal aortic occlusion is contemplated. If the latter is not necessary, rewarming can be started at any time. Any other method of inducing hypothermia has serious drawbacks and should not be used, particularly in view of the fact that in the majority of cases the aneurysm does not reach the renal arteries.

Blood Administration.—The administration of blood is always necessary in these cases, but surgery should not be delayed in the hope that blood transfusion will improve the patient's condition.

The requirements for rate and amount of transfusion can be related to the three different stages in the operation.

1. *Before Operation and in the Early Stages of Laparotomy.*—The blood-pressure is usually very low, the systolic blood-pressure immediately before operation in the present four cases being 60, 50, 70, and 50 mm. of mercury respectively. The amount of blood transfused in this period should be small, as it is felt that any increase in blood-pressure will tend to increase bleeding. In the above cases an average of 3 pints of blood were transfused during this phase.

2. *After Application of the Aortic Clamp.*—When the aorta is clamped there is a sharp increase in both systolic and diastolic pressure due to the sudden increase in peripheral resistance (*see Fig.* 302). Thereafter, over about 30 mm., the blood-pressure falls to about the previous level. This is probably due to the marked diminution in venous return to the heart.

At this stage, massive transfusion may be dictated by the patient's condition, and if it is necessary, it is best carried out into the aorta above the clamp. However, the problem is generally less acute. Although blood-loss from the effective circulation during this phase is generally minimal, nevertheless in *Cases* 2 and 3, 4 and 5 pints were required respectively to stabilize the blood-pressure.

There is no doubt that the amount of blood required at this time is difficult to judge and there is a tendency to overtransfuse. During this phase when the aortic clamp is in situ and insertion of the graft

proceeding, the blood-pressure can be stabilized, preferably at a level somewhat below the estimated normal (*see Fig.* 302).

3. *On Release of Aortic Clamps.*—When the clamps are removed from the aorta and vessel to one leg there may be a sudden fall in blood-pressure. This is probably due to a sudden diminution in peripheral resistance, reflex hyperæmia of the lower leg vessels, and varying degrees of hæmorrhage from the suture lines. It is advisable to reserve at least two pints of blood for this stage in case hæmorrhage is excessive. In the absence of hæmorrhage, dangerous falls in the blood-pressure may be checked by pneumatic tourniquets on the legs (Kenyon and Cooper, 1956). They have not been used in these present cases.

The fall in blood-pressure is followed by a gradual rise to normal levels as the vascular tone of the limb recovers and as venous return from the leg increases. This fall and rise is repeated when blood is allowed to flow to the second leg, but the changes are smaller in degree (*see Fig.* 302).

OPERATIVE MANAGEMENT

Consideration has been given in the past to the question of whether proximal control of the aorta by opening the chest is necessary (Javid, Dye, Grove, and Julian, 1955). The advantages of early control of the hæmorrhage are obvious, and proximal control of a major artery is usually worth while in arterial surgery. However, the problem here is more complex. We believe in retrospect that thoracotomy was unnecessary in *Case* 2. In the event, it imposed a considerable burden on the patient because of the gross degree of emphysema present and added complications which caused his early post-operative death, although a fatal outcome would have occurred later because of the gangrenous sigmoid colon. Javid and others reported their experience in treating ruptured aortic aneurysms, and in 2 out of 4 cases thoracotomy was carried out to obtain control of the thoracic aorta. In both cases the patient died. It is of interest that in each case the tear in the aneurysm was at the bifurcation area. With finger dissection mobilization of the aorta below the renal arteries is safe and easy, and this technique, rather than a more definitive procedure, makes it possible to burrow round the aorta through the blood-clot. Furthermore, clamping the aorta for even a short time above the renal arteries will increase the risk of renal failure, which is always a serious hazard. It may be added that when the aorta is clamped below the renal arteries, the risk of renal failure immediately starts to diminish. In most cases we feel that thoracotomy is unnecessary. Gerbode (1954) also believes that it is best avoided. Where it is necessary because of upward extension of the aneurysm or the need for renal artery, reconstruction hypothermia should be employed.

In all four cases an aortic homograft was used, and this is to be preferred if it is available. It is true that there are probably physical advantages in using a prosthesis (Dale and Mavor, 1958), but it is our opinion that these are outweighed by the advantages inherent in the use of the homograft. The latter include the absence of hæmorrhage both from the anastomoses and the graft itself. Where in most cases blood-loss has already been excessive and the peripheral circulation only recently stabilized, this is an important consideration. The fact that in most cases of aneurysm the diameter of the proximal aorta is always larger than that of the homograft is not a practical objection, as by tailoring the end of the homograft a safe anastomosis can be obtained. The advantage of the homograft is more apparent at the distal reconstitution, where the anastomosis is best made end-to-side to the external iliac artery, as the latter is usually healthy and easy to mobilize. There is some evidence, too, that even in large arteries this type of anastomosis has advantages as regards long-term patency over the end-to-end (Linton and Menendez, 1955; Cockett and Norman, 1958). Furthermore, its use greatly simplifies restoration of blood-flow through the internal iliac arteries, a matter of some importance, especially on the left side, where with destruction of the inferior mesentery artery and left-sided extravasation, the blood-supply of the sigmoid colon may be critical.

Heparin was not administered systemically, but was used locally in all anastomoses and injected distally in a considerable volume of saline into the femoral arteries. This is an important procedure which should not be neglected. In *Case* 3, because of weakness of the right femoral pulse which was noted pre-operatively, the external iliac artery was opened just above the inguinal ligament, and a considerable amount of mural clot adherent to the posterior wall of the vessel was removed both proximally and, to a less extent, distally. Some clot was also removed from the left side through a similarly placed incision. It is advisable to be certain that the whole length of each external iliac artery is clear of clot, arteriotomy above the inguinal ligament being particularly indicated in cases where, pre-operatively, the femoral pulse is noted to be weak.

In each case only the anterior wall of the aneurysm was removed. No attempt was made at resection, as the posterior wall was firmly adherent and dissection risked serious damage to the great veins. A great deal of clot was scooped out of the aneurysmal sac, but much was left adherent to the wall and in unopened common iliac aneurysms. A cuff of aorta below the renal arteries was prepared by dividing the posterior wall of the aneurysm at this point and dissecting it up superiorly. It may be necessary to mobilize and divide a pair of lumbar arteries. Elsewhere, bleeding usually did not occur from the orifices of the lumbar arteries, but they require to be inspected and sutured if necessary. When in position the graft lay inside the aneurysm. This was far from ideal, as the homograft is given no support from viable host tissues. However, in *Case* 3, the only long-term survival, no complications resulted. In *Case* 4 the post-mortem findings revealed that for a period the homograft may be surrounded by a pocket of liquefied debris, which presumably eventually inspissates. Except at the suture lines, which become firmly fibrosed, the homograft will thus remain unchanged until the host blood-supply reaches it. Concerned about the lack of support for the homograft, Cooley (1955) advised suturing the greater omentum around it. This, however, will prevent closure of the posterior peritoneum and must increase the danger of small-bowel obstruction in the post-operative phase when ileus will be present for several days.

COMMENT

The prognosis in the untreated abdominal aortic aneurysm is uniformly bad, and in most instances this is related to the complication of rupture. The contributions of Colt (1927), Kampmeier (1936), Estes (1950), and Barratt-Boyes (1957) indicate that rupture is the cause of death in about three-quarters of all cases, and that it occurs usually within a few years of diagnosis. In most instances the prognosis is much worse than this and it is possible for an aneurysm to develop, reach a large size, and rupture in the space of a few months (*Case* 4). To some extent this may explain why, in our experience, few cases of abdominal aortic aneurysm are seen before rupture. Over the 3-year period covered by this series, only 2 cases were seen in which the patient was symptom-free, while there were 10 cases (in addition to the 4 reported here) in which the patient died in hospital as a result of the complication of rupture. In these, for various reasons, surgical treatment was not carried out. It is apparent then that in the treatment of the majority of cases the most important single step is to make the diagnosis of rupture at an early stage, so that surgery can be undertaken with the patient in as good a condition as possible, a circumstance which will have a direct bearing on the outcome of such treatment (*Table I*).

Table I.—Diagnosis and Outcome in 4 Cases of Ruptured Aortic Aneurysm

Case	Age (years)	Duration of Rupture	Duration of Collapse	Outcome of Surgery
1	58	17 d.	4 hr.	Operative death (cardiac arrest)
2	65	38 hr.	2 hr.	Died 4th post-operative day
3	63	10 hr.	1½ hr.	Recovery
4	68	3 d.	14 hr.	Died 3rd post-operative month

The symptomatology of rupture is not as yet widely recognized. Barratt-Boyes, who recently described the clinical picture of rupture, found that in only 7 out of 18 cases was the correct clinical diagnosis made. Abdominal pain is the most characteristic finding. In this respct the patient may have had persistent backache for some time. This should always be taken as a serious sign. It indicates that the aneurysm is enlarging, and even more important, that rupture is imminent. When rupture occurs, severe pain develops. Typically, the onset of this pain is sudden. Its situation varies, but it is usually in the back, often to the left, and it may radiate as a result of lumbar root irritation. In this way it may resemble the acute prolapse of an intervertebral disk. The onset of such sudden persistent pain should always be taken as an indication that the aneurysmal sac has ruptured. Later, the pain becomes intermittent, so that there may be intervals when the patient is free of complaints. This period of apparent well-being may last for several days or even, on rare occasions, for weeks. Usually, as already stated, it lasts about 12 hours (Barratt-Boyes, 1957). As a cause for the delay in progression of symptoms or for partial recovery, it has been suggested that the tear in the aneurysm may become temporarily plugged with clot (Gerbode, 1954). It would also seem that the loss of blood in the retroperitoneal tissue may proceed comparatively slowly because of the increasing pressure within the hæmatoma. Inevitably pain recurs, becomes more severe and more widespread, depending on the main area of blood extravasation. Shock now develops and its occurrence should always suggest the diagnosis. Peripheral collapse and the signs of shock may thus be absent for a long time, and it cannot be overstressed that in the initial stages the illness is usually not a dramatic one. Nevertheless, this is the time at which the diagnosis must be made if the patient is to have a real chance of recovery. Early diagnosis is difficult in those cases in which a pulsating abdominal mass is not located, presumably because the aneurysm is small. Where the aneurysm is palpable, as it usually is, the diagnosis is simplified. In certain cases the aneurysm may rupture into the gastro-intestinal tract, the duodenum being involved in about 75 per cent of all reported instances. Even with such a complication, however, there is ample interval for surgical intervention (Voyles and Moretz, 1958). By the time extravasation is widespread, abdominal signs are pronounced, with tenderness, guarding, and distension resulting from blood in the juxtaperitoneal tissues and perhaps in the peritoneal cavity itself. These abdominal findings may make palpation of the aneurysm difficult.

The development of shock is a dramatic event, and as seen from the above cases, a surgical opinion is not generally sought until it is well established. In many instances surgical treatment will by then have little chance of success. The sudden onset of collapse and shock is probably due not so much to any sudden increase in hæmorrhage, but to the inevitable peripheral vascular decompensation which will occur in the face of continued bleeding. Clinically little is gained by suggesting that the aneurysm may leak before it ruptures, and this trend of thought may suggest that some delay in surgical treatment, perhaps to transfuse the patient, is justifiable. In fact it seems more likely that when pain occurs, the aneurysm has ruptured, and the duration of the final illness depends entirely on the size of the rupture. In such circumstances, surgery is imperative at the earliest possible moment. In *Case* 1 it was not fully realized that the aneurysm had already ruptured when the patient was first admitted to hospital. Delay, firstly in carrying out needless investigations and, secondly, in organizing surgical treatment resulted in the patient reaching the operating theatre in the worst possible condition. Blood replacement had little effect in improving this, and the outcome of surgery under such circumstances was not unexpected. Although there have been instances where small ruptures have healed spontaneously, and the patient lived for several years to die subsequently of a major tear (Estes, 1950; Barratt-Boyes, 1957), this event occurs so rarely that it cannot be allowed to influence surgical management.

Even after successful reconstruction of the aorta there are many post-operative complications which can arise. Technical failure at an anastomosis is rare, particularly when a homograft is used. Infection is a

great danger, but the presence of debris and blood-clot in the retroperitoneal space does not make it inevitable. Appropriate treatment must always anticipate intestinal ileus, which generally attends aortic surgery, and in cases of rupture is likely to be marked. Renal failure represents one of the more serious complications, and Shumacker (1955) lost 2 out of 5 patients from this cause. Its incidence is largely dependent on the period of pre-operative hypotension, which can only be curtailed by earlier recognition of the symptoms of rupture. It has already been noted that clamping of the aorta above the renal arteries, when this is done at normal temperatures, will add to the risk.

Recovery of the lower limbs from the acute ischæmia of operation is usually uneventful and it would seem that marked ischæmia can be tolerated for many hours. However, the risk of arterial embolism or acute thrombosis must be considerable, and when gangrene of a limb occurs, it probably follows such a complication. It is interesting that there may be a considerable delay, even of days, in the return of adequate peripheral circulation. In *Case* 3 steady improvement in the peripheral circulation was noted each morning, suggesting that during sleep release of excessive vasoconstrictor tone in the small vessels was occurring.

Peculiar to this operation is the complication of ischæmic gangrene of the sigmoid colon, and it represents a serious hazard in all left-sided aneurysmal sac ruptures. There seems little doubt that the additional factor of extravasation is responsible for the blood-supply of the sigmoid colon being so critical. In the treatment of uncomplicated aneurysm, no complications have followed ligation of the inferior mesenteric artery and failure to reconstitute both internal iliacs (Bahnson, 1954). In the ruptured aneurysm, it is always wise to reconstitute the internal iliac arteries, or at least the one on the left side. Nevertheless, even when this is done, as in *Case* 2, sigmoid gangrene may result, although in this instance post-operative hypotension may have been a contributory factor. Dale (1955) experienced a case where ischæmic gangrene of the sigmoid colon resulted in peritonitis and death on the fifth post-operative day. Although one of the successful resections reported by Javid and others (1955) followed a left-sided rupture, it would thus appear likely that left-sided rupture has a worse prognosis than right.

The mortality in this form of emergency surgery may always be considerable. Recent figures (Javid and others, 1955; Shumacker, 1955; Farrar, Adson, Kirklin, Martin, and Barker, 1956) suggest that only about half the patients can be saved. Cooley (1955), however, reported 11 patients surviving out of 18. There may always be a proportion of operative deaths due to cardiac arrest, for the operation is a formidable one. A more limited first-stage procedure, such as patching or suturing the tear, is generally not feasible except from inside the aneurysmal sac, and if the operation has proceeded this far, aortic reconstruction should be undertaken. The answer to reduced mortality lies in increased technical experience of the operation and, above all, in earlier diagnosis and more urgent surgery.

SUMMARY

Four cases of ruptured aneurysm of the abdominal aorta are described in which reconstructive aortic surgery was undertaken. Only one case had a successful outcome.

Some points in anæsthetic and operative management are discussed.

Stress is laid on the need for early recognition of the symptoms of rupture. Once the diagnosis is made, immediate surgery is indicated.

Mortality is considerable, but should decrease with greater awareness of the need for operation.

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HYDATID DISEASE OF THE SPINE AND ITS NEUROLOGICAL COMPLICATIONS

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HYDATID disease in man was common enough in Europe at the beginning of the century, but within recent years it has been assumed that the disease only occurs with any frequency in the more temperate zones of the southern hemisphere. Perhaps this is because the last comprehensive account was by Dew and Ivanissevich, 1934). Recent literature is scanty, but Woodland (1949) reported 6 cases from Sydney. All had paraplegic symptoms and there was only one long-term survival. Berkay (1954), from Turkey, described 2 cases with paraplegia which improved after operation. Hamman and Joubert (1957), from

FIG. 306.—*Case* 1. Lateral radiograph showing hollowed-out area in body of L.4. There is increased density of the rest of the bone and it has slightly collapsed.

FIG. 307.—*Case* 1. Anteroposterior radiograph and air myelogram. L.4 is slightly compressed and the thecal shadow is narrow at this level.

in 1928 from Australia. The disease is universal, and it tends to become a serious public health problem when the ratio of sheep to man in a country exceeds 0·4 (Whitten, 1958). With intensification of agriculture in a hungry world the disorder cannot be neglected. The Registrar-General (1957) records 152 deaths from hydatid disease in the years 1946–56 inclusive from England and Wales. New Zealand statistics (Foster, 1958) indicate that the average percentage of deaths to new cases is about 16, so that there should be 950 new cases for this period. Thus Barrett and Thomas (1952), from unstated British sources, collected 71 cases of pulmonary hydatid disease over 14 years, and the lungs are affected in only about 10 per cent of cases of hydatid disease (Dew, 1928).

Bones are involved in about 1 per cent of cases, of which about half will be in the vertebral column with the frequent sequel of paraplegia (Dew, 1928, South Africa, had 6 spinal cases. All had paraplegia and only one had spinal symptoms for longer than 7 months, which is unusual. Immediate benefit was obtained from laminectomy in most. The difficulties in diagnosis may be considerable, as a recent personal case showed. Two other patients have been treated in the Department of Neurosurgery, Dunedin Hospital, since 1943, and the opportunity is taken of re-defining the syndrome of spinal hydatid disease.

CASE REPORTS

Case 1 (NS 7680).—A boy aged 14 years was admitted to the Neurosurgical Unit on April 14, 1958. At the end of 1954 he had had low backache and pain in the left knee. He was admitted to Wanganui Hospital where he improved on bed-rest; subsequently he had had intermittent attacks of backache and pain in the left hip which had remitted on rest. In January, 1958, he had got much worse and was re-admitted to hospital. He was found to

have a stiff lumbar spine and reduction in the range of the left hip movements. Both knee-jerks were depressed. Over the years many radiographs of the lumbar spine had been taken and there was a slowly progressive destruction of the body of the fourth lumbar vertebra (*Fig.* 306). A lumbar puncture gave a fluid with a protein of 65 mg. per cent and an equivocal response on jugular compression. An air myelogram showed narrowing of the theca at L.4 (*Fig.* 307).

ON EXAMINATION.—The lumbar spine was rigid with a scoliosis to the right. There was little tenderness, but

FIG. 308.—*Case* 2. Anteroposterior radiograph and myelogram. There is destruction of the inner end of the right eleventh rib and transverse process. The twelfth rib and transverse process are partly eroded. Some calcification in a paravertebral mass is seen between 10th and 11th ribs on right. Myelogram shows a block at T.11–12 level with displacement of the column to the left and filling defects above.

percussion of the L.4 spine was uncomfortable. The range of the left hip movements was slightly reduced. The patient had no weakness or wasting of the legs and sensation was normal. Both knee-jerks were much reduced and the ankle-jerks were brisk. There was no pain on jugular compression.

Mantoux reaction was doubtfully positive to the second strength. Casoni and hydatid complement-fixation tests were negative. Radiography of the chest was normal. The radiographs of the spine were reviewed. There was slight collapse of the body of L.4. There was a hollowed area in the middle of this body with slight increase in the density of the surrounding bone. The pedicles were a little enlarged (*Figs.* 306, 307). This lesion was just visible on the first radiograph, taken in October, 1954, and had slowly increased since. There was no paravertebral shadow. A tentative diagnosis of a benign destructive lesion, possibly tuberculous, was made. As the future management of the patient depended on a more precise diagnosis, exploration was advised.

AT OPERATION.—On April 16 a laminectomy of L.4 was done. The paravertebral muscles were normal. The arch of the lamina was thin and as the bone removal was carried towards the pedicles a bluish mass was seen on the left side. This was separate from the dura. When the theca was retracted to the right more of the mass became visible, and it seemed to be arising from the back of the body of the vertebra. It was first thought to be a hæmangioma and a hollow needle was inserted. Clear fluid came back into the aspirating syringe and some small white specks could be seen floating in this fluid. These were the brood cysts of a hydatid cyst, and this was confirmed by a microscopical examination. The cavity of the cyst was filled with 10 per cent formalin solution. The capsule of the cyst was then removed in one piece. Inspection of the cavity showed that the hydatid cyst had occupied the centre of the body of L.4 and had embraced the theca in a horseshoe fashion. There was no sign of any daughter cysts. The local tissues were swabbed with more formalin solution. The wound was closed in layers.

PROGRESS.—Recovery was rapid and the patient left hospital on May 2. He was seen at follow-up on June 11, 1958, when his spine was mobile and the knee-jerks were brisk.

PATHOLOGY (Dr. R. Rodda).—Fluid from the cyst contained numerous hydatid scolices. The specimen was a collapsed cyst 7 cm. in maximum dimension, which was a hydatid cyst histologically.

Comment.—The apparent solitary cyst in this case is unusual and this can be confirmed only by the passage of time. While tuberculosis seemed a likely diagnosis, the Mantoux reaction was negative until the patient's last admission to hospital. If Whalley's (1958) article had been available, an osteoclastoma would have been the most favoured diagnosis. The long history had at least suggested a benign lesion. The patient was young to be suffering from hydatid disease.

Case 2 (NS 330).—A woman aged 45 years was first admitted to the Neurosurgical Unit on June 26, 1944. She was complaining of low thoracic backache for six months and pains in her legs for four months. The pains were made worse by any movement and she had been bedridden for three months. In 1941 she had had pain in the side of the right thigh and this had gone after a year, but the area had remained numb.

In 1924 she had developed pulmonary hydatid disease, for which thoracotomies were done in 1928 and 1936. Laparotomies were done in 1941 and 1943 for hydatid disease of the liver. Her condition had deteriorated since this last operation.

ON EXAMINATION.—She was a frail woman with a blood-pressure of 200/130 mm. Hg. She had a marked weakness of the right leg and a lesser weakness of the left leg, but without wasting or alteration of tone. There was loss to pin and cotton-wool over the outer side of the right thigh. The right ankle-jerk was just present and that on the left was absent. Plantar responses were extensor. Her back was rigid and there was a bulge to the right of the spine at T.12 level.

Hydatid complement-fixation test and Casoni test were positive. The leucocyte count showed 12,050 cells per c.mm. and 3 per cent were eosinophils. The cerebrospinal fluid contained 180 mg. per cent of protein and there was a manometric block.

Radiographs of the chest showed a round mass in the right middle chest. There was erosion of transverse process and pedicle on the right side of T.12 and this involved the inner end of the twelfth rib. The inner end of the eleventh rib was destroyed. Some calcification could be seen in paravertebral tissues. The patient had a scoliosis to the right. A myelogram by the cisternal route showed a complete block at T.11/12 level with displacement of the column to the left (*Fig.* 308).

AT OPERATION (Mr. M. A. Falconer and Mr. J. A. Jenkins).—A hemilaminectomy of T.10-12 on the right was done. The spinal canal contained many extradural hydatid cysts up to 1 cm. in diameter for the whole length of the exposure, and they were removed. The inner end of the twelfth rib was loose and when this had been excised a collection of hydatid cysts above and below the diaphragm in the paravertebral gutter was entered. They communicated with the spinal cysts by way of the intervertebral foramina. The mass was removed and the area cleaned with 2 per cent formalin solution. The dead space was packed with petroleum jelly gauze and the wound was closed with the gauze as a drain.

PATHOLOGY.—There were numerous large hydatid cysts and daughter cysts which had the typical histological appearances.

PROGRESS.—The gauze was removed on the third day. The patient left hospital on Aug. 23, 1944, when she was walking and the wound was healed. Both ankle-jerks were absent. There was no manometric block on lumbar puncture.

Second Admission.—She was admitted on Nov. 28, 1945. The backache had returned in the previous six months. There were no abnormal neurological signs, but there was swelling of the back to the right of T.12. The radiographs were unchanged. On Dec. 31 Mr. Falconer and Mr. S. Wilson excised a mass of hydatid cysts, 8 × 5 cm., from the right paravertebral region. The patient left hospital on Jan. 17, 1946.

Third Admission.—This was on Sept. 3, 1947. In October, 1946, further hydatid cysts were removed from the back in another hospital. Since then the patient had had pains in her legs, and recently some difficulty with micturition had occurred. There was weakness of both legs which was more marked on the right. The plantar responses were flexor. Radiographs of the spine indicated subluxation of T.12 with kyphoscoliosis. There was more erosion of the bodies of T.11 and 12 (*Fig.* 309). There was a complete block on lumbar puncture and the cerebrospinal fluid contained 550 mg. per cent of protein.

On Sept. 19, Mr. A. James and Mr. J. A. Jenkins reopened the laminectomy wound. There was a mass of cysts compressing the theca at T.11 and these extended into the paravertebral gutter. The theca was very angulated over the collapsed vertebra. The area was cleaned out and the patient was much improved by this operation.

As the spine was thought to be unstable, on Nov. 7 an onlay spinal graft was done, using the tibia as a donor site.

Progress.—The patient was transferred to a private hospital on Dec. 19, 1947, very much improved and with minimal pain. There was a slow recurrence of her symptoms and she became a morphine addict. She died in October, 1952, and there was no autopsy.

Comment.—This is a typical case of hydatid disease of the spine with a triple abscess and paraplegia. While the patient had had pulmonary hydatid cysts removed it is extremely unlikely that the spinal disease was the result of secondary invasion by paravertebral residues. At no stage was cure of the spinal disease possible.

Case 3 (NS 85).—A woman aged 20 years was admitted to the Neurosurgical Unit on Sept. 4, 1943. In February, 1942, she had had a minor injury of her right buttock which was followed by local pain, and in ten days there was a right sciatica. This lasted for three months, when she was put to bed. After three months' rest her symptoms subsided. She had another fall in April, 1943, which gave her buttock and perineal pain, and this improved on rest. She now noticed numbness of the right buttock and back of the thigh. The pain became worse during August and she had urgency of micturition.

ON EXAMINATION.—There was marked wasting of the right buttock, hamstring, and calf muscles with associated weakness. She had anæsthesia to pin and cotton-wool over the right saddle area and back of the thigh. Knee- and ankle-jerks were present and equal. Straight leg raising was to 30° on each side. The lumbar spine was rigid and flat. The right sacro-iliac area was tender.

Radiographs of the chest and lumbar spine were normal. Hydatid complement-fixation test (post-operative) was negative. There was no block on lumbar puncture, and the cerebrospinal fluid contained 40 mg. per cent of protein. A myelogram showed a filling defect on the right side of the terminal sac and this appeared extradural (*Fig.* 310).

AT OPERATION (Mr. M. A. Falconer).—A laminectomy of L.5 and S.1 and 2 was done. A large unilocular cyst was found in the extradural space of the spinal canal which had eroded the body of the sacrum. The cyst was punctured in removal and the excision seemed complete.

PATHOLOGY.—Unilocular cyst, 5 × 2 × 2 cm., whose layers corresponded to those of a hydatid cyst. No scolices were seen.

PROGRESS.—The patient left hospital on Oct. 6, 1943, with the wound healed, she was free from pain, but there was no change in the neurological signs.

Second Admission.—She was re-admitted on Dec. 10, 1945. Three weeks before she had developed pain and swelling in the region of the incision. There had been a fever to 101° F. Examination showed a tender swelling of the scar which spread out into the right buttock. There was no muscular wasting, but the previous anæsthesia was still present. Radiographs of the lumbar spine and pelvis showed changes consistent with the previous myelogram and laminectomy. On Dec. 23 Mr. Falconer and Mr. J. A. Jenkins opened the area and came upon a mass of infected hydatid cysts arising from the sacrum. The area was cleaned out and the wound closed over a gauze drain. *B. proteus* was grown from the infected material. The patient was able to go home on March 6, 1946, with a slight discharge from the wound.

FIG. 309.—*Case* 2. Anteroposterior radiograph four years after *Fig.* 308. There is partial collapse of T.11 with a kyphoscoliosis. Inner ends of 11th and 12th ribs and transverse processes have disappeared. Paravertebral calcification is still present.

Third Admission.—The patient was again admitted on March 8, 1949. The previous sinus had soon healed and she had been well until the middle of February, 1949, when the sinus opened and some cysts had appeared. On April 1 Mr. Falconer and Mr. Jenkins saucerized the whole area and the main part of the infection came from the bodies of the upper sacral vertebræ. A sinogram later showed a tract leading into the body of S.2 (*Fig.* 311). She left hospital on June 14 with the sinus not yet healed.

Progress.—The sinus continued to discharge and the patient was in hospital for two days in August, 1949, when Mr. James laid part of the sinus open again. After this it healed up. She married, and had a baby in 1956. She was seen in the out-patient department on April 24, 1958,

when she was complaining of pain in the back of the right leg. The scars were well healed. The sole neurological finding was anæsthesia of the right buttock and back of the thigh. Radiographs of the lumbar spine and pelvis indicated no change in appearances since 1949.

Comment.—It is hard to know whether in reality this was a solitary cyst, as it was punctured in removal and the wound was not disinfested with formalin solution. If it was a single lesion it probably arose from the body of

FIG. 310.—*Case* 3. Myelogram showing filling defect at L.5 from the right.

S.2 and spread up the spinal canal. The occurrence of a low-grade surgical infection seems to have produced a lasting cure.

DISCUSSION

Pathology.—The classical account of the pathology of the hydatid cyst in the liver and lungs of man was given by Thomas (1884). Dew (1928) and, recently, Barrett and Thomas (1952) further described these lesions and they will not be discussed again here. The lodgement of the parasite in bone happens in about 1 per cent of all cases of clinical hydatid infestation. Ivanissevich (1934) collected 81 cases from the Argentine and gave a good account of the condition. His estimate of involvement of the spine in 50 per cent of bony cases is agreed to by most authors. Grisel and Dévé (1929) fully described the vertebral form of the disease which in France is called *le mal de Pott hydatique*.

In most tissues the hydatid cyst is single, but may within itself form daughter cysts by a process of endogenous loculation. Multiple cysts are the rule in bone, but they are formed by extrusion and called exogenous loculation. The larval stage of development begins in the bone-marrow and the initial cyst excites only a minimal cellular reaction, so that no adventitia is formed outside the laminated membrane or ectocyst. As the cyst enlarges it insinuates itself into the interstices of the marrow, and in so doing small loculi are formed. These become separated from the parent cyst and the end-result is a mass of small cysts which contain little fluid and have been likened to grains of boiled sago. By relentless progression the medullary

FIG. 311.—*Case* 3. Anteroposterior radiograph of lumbar spine and pelvis and sinogram. The theca is outlined and is displaced to the left by the sacral mass which is partly outlined by the sinogram. This is at S.2.

cavity of the bone becomes filled with cysts and eventually the dense cortical bone is eroded away. Some trabeculæ remain so that the bone appears to consist of large loculi. The articular cartilage and periosteum are invaded and once the soft tissues have been reached large hydatid cysts are formed. The bone becomes weak and pathological fracture occurs. It is unusual for a solitary bone-cyst to be formed. Suppuration in bony hydatid disease rarely happens except as a result of surgical intervention. The disease is insidious and chronic.

The analysis of Grisel and Dévé (1929) indicated that the vertebral form of hydatidosis is like that in bone elsewhere, the closeness of the spinal cord adding a complication. A few patients will have hydatid disease in clinical form elsewhere in the body, but it is an event of great rarity for a vertebra to be invaded from the paraspinal tissues, so that extraspinal spread from a primary vertebral focus is the usual method of progression. The body of the vertebra is the common site for the disease to start in and it is unusual for three or more adjacent bodies to be affected. The body becomes a hollow shell filled with small cysts which infiltrate into the pedicles and lamina. Sometimes the primary site may be in the lamina without the body being involved. The intervertebral disk is spared and this is a characteristic of the disease. It is remarkable

how in spite of great destruction of bone the shape of the body is maintained until late. A severe degree of kyphosis or gibbus occurs in 10 per cent of cases.

The distribution of the lesions at the different vertebral levels in percentages is of the order cervical 10, thoracic 50, lumbar 20, and sacral 20. The thoracic spine from T.6 to T.12 bears the brunt of the disease with a maximum incidence at T.12. If the sacrum is regarded as one vertebra then it would be the commonest site for the disease.

Spinal hydatidosis rarely remains localized and invades the paravertebral tissues. As any part of the vertebra may be affected, and the process is not restrained by the periosteum, widespread anterior and posterior infiltration occurs. The anterior extension tends to be one-sided and it is uncommon for a midline symmetrical hydatid abscess to be produced. In the thoracic area the transverse process and head of the rib are invaded and destroyed. A large mass may collect in the paravertebral gutter, but the pleura and lungs are not usually invaded directly. When the abscess extends backwards a single cyst results and the microcystic form here is the consequence of surgical intervention. The combination of hydatid cysts in the vertebra with anterior and posterior extensions is called a triple abscess of which parts may calcify. Abscesses as big as a baby's head have been described.

The spinal canal is infiltrated, but the dura mater always remains intact. The loose extradural tissue is ideal for the dissemination of the microcysts. Primary extradural hydatid cysts have been described, but usually they have arisen from an undetected bony focus. An intradural hydatid cyst is rare and Diez (1945) could only find 8 in the literature. Once the extradural space has been invaded it is only a matter of time before a paraplegia is produced by pressure on the spinal cord. Details of the changes in the spinal cord are not much recorded, but they seem to be the same as in any other compression paraplegia. At least 25 per cent of cases become paraplegic, and this is least common with the lumbar and sacral locations of the disease.

Clinical.—Males are slightly more affected by spinal hydatid disease than females. The occurrence of the disease in women is accounted for by the intrusion of the dog into the domestic situation, and children are similarly infected. The incidence is greatest between 10 and 50 years of age, with the peak in the third decade of life, so that it is least common in children and in the elderly.

The clinical features of spinal hydatidosis are characterized by great chronicity and the disease may be far advanced before the patient seeks medical attention. So long as the parasite is confined to the bone, symptoms are few and severe pain is unusual. Once extra-osseous spread has happened then there is a syndrome of spinal deformity, paravertebral tumour, and paraplegia. The spinal deformity is a kyphosis or kyphoscoliosis, but this is not marked and a gibbus is exceptional. The spinal movements are well preserved and local tenderness and muscular spasm tend to be insignificant. An anterior abscess, except in the neck, is not likely to give pressure symptoms until it is very large. The posterior extension produces a paravertebral tumour which is painless and without skin reaction or fever. When big it is fluctuant and transillumination is possible. Pressure on a lumbar cyst may give pain in the groin and leg due to transmitted pressure upon the cauda equina.

The paraplegia does not possess any intrinsic features which distinguish it from other varieties of spinal cord compression. Radicular symptoms occur first and may be the sole manifestation of lumbar and sacral hydatid disease. The paraplegia is slow in evolution and liable to remission, permitting resumption of normal work. However, a not uncommon sequence of events is for a patient to have had the vertebral disease for years and then to die rapidly with a paraplegia of acute onset. The paraplegia is not usually the first symptom of hydatidosis of the spine.

Radiology.—The radiological appearances reflect the quiet nature of the necrosis of the bone which is the feature of the pathological changes. The body of the vertebra shows a hollowed-out area which may affect most of the bone, but cortical and periosteal reactions are minimal. The shape of the body has been likened to a coffee-bean by French and South American observers. Oblique projections and tomographs may show that the erosive process has spread into the pedicles, transverse process, lamina, and head of the rib. Collapse of the bone only occurs late. When the disease has extended into the paravertebral tissues its shadows can be seen easily, and here there may be some calcification of the cyst walls which will be visible radiologically. Myelography will show the level of the spinal block, but it may be at a different level to that of the bone disease owing to the migration of the cysts in the extradural space.

Investigations.—There are two tests which are specific for hydatid disease in man, and these are the Casoni skin test and the hydatid complement-fixation test. The latter is the more sensitive of the two and false negatives are rare (Bensted and Atkinson, 1958). However, in areas where hydatid disease is endemic a positive result indicates infection, but this may not be relevant to the clinical condition under consideration. A negative result to either or both tests does not exclude the disease. The leucocyte count is not elevated, but there may be an increased proportion of eosinophils.

Differential Diagnosis.—While hydatid disease of the spine has to be distinguished from the many other kinds of bone tumour, those of greatest importance are tuberculosis, osteoclastoma, hæmangioma, and malignant disease. A correct interpretation of the radiographs is often the crucial point in making the right diagnosis.

It is spinal tuberculosis which most closely resembles hydatid disease, as paravertebral tumours may be present in both conditions. The features in favour of tuberculosis are a relatively younger age-group, pain and fever, some bony reaction to the disease, and involvement of the intervertebral disk. Paravertebral calcification occurs with both diseases. It is rare for the head of the rib and the lamina to be affected and for a paravertebral abscess of any size to occur with tuberculosis. A triple abscess is rare and a gibbus common in tuberculosis. Aspiration of a posterior abscess in hydatid disease will give a water-clear fluid and the scolices can be identified under the microscope. The mode of onset

of the paraplegia is not helpful in the differential diagnosis.

Osteoclastoma of the spine is an uncommon lesion and Whalley (1958) has reviewed the literature. This tumour produces a quiet destruction of the vertebral body, which may include the rest of the bone, with ultimate collapse and paraplegia. The radiological appearances are very similar to those of hydatid disease. It may not be possible to be sure of the diagnosis without biopsy.

Hæmangioma of the vertebra is one of the commonest tumours of the spine (Coley, 1949). The clinical features are not very marked and are slow in evolution. The radiological changes are characteristic as there are vertical striations of the bone with less dense areas in between. All the body is involved, but before collapse occurs it is widened. If the pedicles and lamina are affected they are enlarged. Paraplegia occurs when the bone collapses. A related lesion is an aneurysmal bone-cyst of the spine (Beeler, Helman, and Campbell, 1957), but here the cyst-like area is surrounded by a rim of cortical bone which is visible on the radiographs.

Malignant disease of the spine is commonly metastatic from cancer of other organs, and when primary it is due to a plasma-cell myeloma (Coley, 1949). In either case the bone destruction may be considerable, but many bones are affected and the primary site is usually evident. The rare solitary myeloma in the vertebra may be hard to distinguish as the bony reaction can be negligible and biopsy may be needed. This sort of lesion is painful and other evidence of myelomatosis in the rest of the skeleton, peripheral blood, and urine should be sought. These malignant conditions have a shorter history than has hydatid disease.

Perineural sacral cysts with their radicular symptoms have a clinical picture like hydatid disease of this area (Abbott, Ratter, and Leinbach, 1957). These sacral cysts cause erosion of the sacrum and widening of the sacral canal. However, the myelogram is diagnostic, for as in hydatid disease a filling defect is produced. With the sacral cysts the medium enters the multiple arachnoidal diverticula that are part of the condition and becomes dispersed into blobs. Hydatid disease is rarely mentioned in the differential diagnosis of sacral cysts, whose diagnosis should be made with caution where hydatid disease is endemic.

Treatment.—It did not take very much surgical experience to show that hydatidosis of bone was not susceptible to surgical cure owing to the infiltrative nature of the microcysts. Only amputation gives any hope of cure, but this is impossible with the spine, so that in most cases surgery is palliative. The rare cases of a single cyst which seem commonest in the lumbosacral region can be treated with a possibility of cure. It will be hard to remove the cyst without opening it, and here the liberal application of formalin solution of up to 10 per cent may reduce the risks of seeding the cysts. The development of a paraplegia is an indication for laminectomy and decompression of the spinal cord. Return of function of the spinal cord is probable, and as the disease is a chronic one, long-term relief may be achieved. It has been well known that a surgical infection of the bone would eventually eradicate the disease if the patient survived. This led Dévé (1935) to suggest that a deliberate infection with an attentuated staphylococcus might be tried, but even with antibiotics the idea has not been attractive.

Prevention.—Statistics from New Zealand have shown that the general mortality from hydatid disease has remained unchanged at 16 per cent over the past thirty years (Foster, 1958). Prevention remains the only way of cure. Thomas predicted in 1884 that the application of the knowledge of the life cycle of the parasite could eradicate it in ten years. The prophecy is unfulfilled.

There is no known drug which will kill the mature worm in the intestinal tract of the dog or affect the larval phase. However, arecoline is a good vermifuge, and if the dog is denied access to sheep viscera then re-infestation cannot occur. These measures can only be effective if the traditional inertia of the farmer and peasant to hygiene can be overcome. Hydatid cysts in sheep livers mean that it is condemned for human consumption. In New Zealand it is estimated that the country loses £1·5 million a year through such wastage, besides the loss of food.

SUMMARY

1. Three cases of hydatid disease of the spine with neurological features are described.

2. The general picture of vertebral hydatid disease in its pathological, clinical, radiological, and therapeutic aspects is considered.

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CYSTIC HAMARTOMAS AND POLYCYSTIC DISEASE OF THE LIVER

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Cystic lesions of the liver are usually asymptomatic and discovered only at autopsy, but the published cases include many of clinical interest; valuable discussions of the subject will be found in the papers of Comfort, Gray, Dahlin, and Whitesell (1952) and Clagett and Hawkins (1946). From the clinical standpoint, cystic tumours and cysts of the liver form a single disease; pathologically they are a heterogeneous group of lesions of diverse origins. They have been classified in various ways by Sonntag (quoted by Montgomery, 1940), Jones (1923), Davis (1937), and Stock (1952). The following list will give some idea of the possibilities: Cystic hamartomas; solitary cysts; polycystic disease; traumatic cysts; cystadenomas; lymphangiomas; biliary angiomas; mucous gland cysts; ligamentum teres cysts; hydatid cysts.

All these conditions may in practice be very difficult to distinguish one from another; this is as true pathologically as it is clinically, for cysts are often uninformative structures. Moreover, the various types are not all mutually exclusive; for instance, some lesions could be classified equally well as solitary cysts or more specifically according to their pathological nature.

The only novelty in our list is the cystic hamartoma; this is a rare but distinctive lesion of infants and is exemplified in our first two cases. We have found only three well-described cases on record. Symmers and Ward-McQuaid (1950) described a boy aged 19 months who presented with abdominal distension due to a firm, partly cystic mass. At operation a large reddish tumour was removed from the lower surface of the liver. The mass (2060 g., 21 × 18 × 8 cm.) was largely composed of cysts from 2 cm. to 12 cm. in diameter. The cysts had no cellular lining and contained pale yellow fluid or gelatinous material. Diagnosis: Cavernous lymphangiomatoid lesion. Packard and Palmer (1955) described a case (*Case* 2) of a boy aged 19 months who presented with abdominal enlargement. At operation a large cystic tumour was removed from the right lobe of the liver. The mass (1550 g., 20 × 15 × 14 cm.) was composed of cystic spaces mainly filled with gelatinous material. The cysts were lined by flattened cells. Diagnosis: Hamartoma. In their *Case* 7 a boy aged 12 months had a large abdomen two-thirds filled with a firm mass that felt cystic. At operation the mass was removed from the right lobe of the liver. The mass (1500 g.) showed many cystic spaces up to 10 cm. in diameter. The walls of the spaces were dense and whitish. Diagnosis: Hamartoma.

CASE REPORTS

Case 1.—**Cystic hamartoma of liver.**

A 3½-year-old male was admitted to the District Infirmary, Ashton under Lyne, on May 30, 1950. The only history obtainable revealed that he had been off colour for several weeks, and had vomited several times two days prior to admission. The family doctor had discovered an abdominal tumour during a routine check-up, and referred the child for specialist opinion. During early April, 1950, he had been an in-patient for circumcision and repair of umbilical hernia; the clinical record made at this time contains no reference to any abdominal mass.

On Examination.—The boy appeared healthy, well nourished, and of average size for his years. No abnormality was discovered on clinical examination of respiratory and cardiovascular systems. A mobile, well-defined, globular mass was readily palpable in the right abdomen. It appeared to be as big as a full-sized grapefruit, and was not tender. It was impossible to get above it under the right costal margin, and it extended well back into the loin, although its posterior limit was difficult to define. It was dull to percussion, and was thought to be renal in origin.

The blood-count revealed a hæmoglobin of 90 per cent, and no abnormal features. Urine examination disclosed a faint trace of albumin and a growth of *Esch. coli* on culture, but no red blood-cells, pus cells, or casts. Radiography of the chest failed to reveal any pulmonary lesion. Excretion urography showed both renal silhouettes to be normal in shape, size, and position. Concentration of the contrast medium was satisfactory and prompt. The right pelvi-caliceal system showed early back-pressure effects due to some kinking of the upper ureter, but no obstruction was seen.

At Operation (R. T. G.).—On June 20, 1950, under general anæsthesia, the tumour was found to arise from the under surface of the right lobe of the liver, lateral to the gall-bladder. Liver substance was stretched out thinly over the cephalic surface of the tumour. It was grey, heavy, smooth-surfaced, elastic, and roughly spherical. It was about 12 cm. in greatest diameter, and there were no adhesions to its surface. A few small fleshy lymph-glands were noticed at the porta hepatis, and one was taken for microscopical examination. Liver tissue close to the tumour appeared normal on palpation. In the belief that this was a primary tumour of liver, resection was undertaken. Doyen clamps were placed lightly on normal liver a few centimetres from the margins of the tumour, and the diathermy knife was used to cut through liver between clamps and tumour. The clamps were removed and spurting vessels were caught and coagulated. Hæmorrhage from the cut surfaces was less than expected, and was controlled by deep catgut sutures. The two cut surfaces of liver were sutured in apposition with gelfoam interposed. The abdomen was closed with drainage. Two pints of blood were transfused during and after operation.

Post-operative Progress.—Recovery was slow but uneventful, and the boy left hospital fit and ambulant on the thirty-seventh post-operative day. He was seen at intervals after discharge. The site of the drain was slow in healing over, and there was some keloid change in the scar. He kept in excellent health, but two years later he died of leukæmia. No post-mortem was obtained.

The Specimen.—This was a piece of liver containing cysts up to 7·5 cm. in diameter (*Fig.* 312). The cysts contained material that after fixation was yellow, opaque, and gelatinous. The inner surface of the cysts was smooth or finely granular. Microscopically the larger cysts had thick walls of cellular fibrous tissue, which was sometimes œdematous. The fibrous tissue was usually lined by a single layer of cells, which were flat (*Fig.* 313), cuboidal, or columnar (*Fig.* 314); in one cyst all three types of cell were seen in direct continuity, the flat cells clearly representing the result of pressure or stretching on the taller cells. The taller cells were indistinguishable from bile-duct epithelium, and there can be little doubt

that the lesion was of bile-duct origin. An eosinophil coagulum—the remains of the cyst contents—was sometimes adherent to the cellular lining. Over some areas the fibrous tissue was unlined and inflamed (*Fig.* 315). There were many cysts of microscopic size; the lining cells were flat, cuboidal, or columnar, as in the larger cysts.

The liver tissue in which the cysts lay was rather fibrous and heavily infiltrated with lymphocytes and

FIG. 312.—*Case* 1. Cut surface of cystic mass from liver.

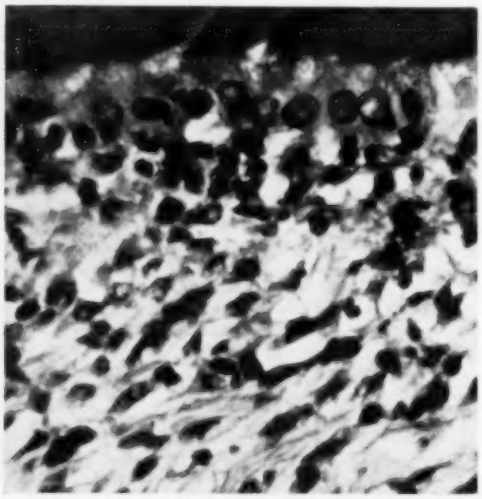

FIG. 314.—*Case* 1. Cyst wall lined by columnar cells. These are covered by coagulum (dark grey). H. and E. (× 536.)

polymorphs. The lymph-gland showed the normal pattern. The germ centres were large and showed mitotic figures. The sinuses were wide and their endothelium was abundant. These appearances are consistent with infection in the region drained. There was no sign of leukæmia.

Case 2.—**Cystic hamartoma of liver.**

A girl aged 10 months was admitted to the Duchess of York Hospital for Babies in March, 1954. For three months the mother had noticed an increasing swelling of the baby's abdomen.

ON EXAMINATION.—A frail, moderately nourished child weighing 24 lb. The abdomen was enormously distended and tense and it was impossible to feel any intra-abdominal structure (*Fig.* 316). The whole of the right side of the abdomen, however, was dull to percussion. Other systems were normal on clinical examination.

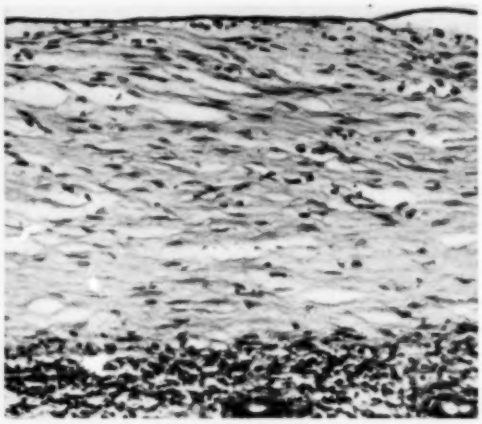

FIG. 313.—*Case* 1. Cyst wall (light grey) covered by flat cells (dark line at top). H. and E. (× 112.)

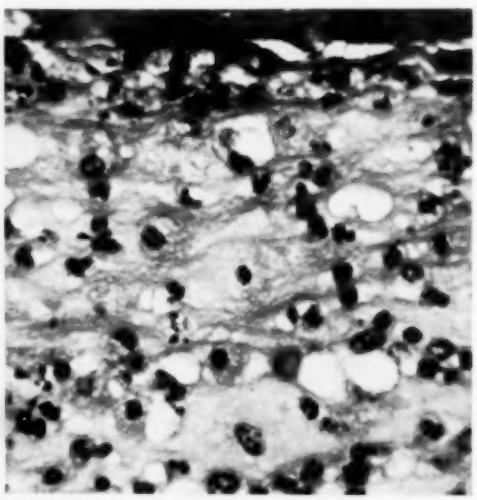

FIG. 315.—*Case* 1. Unlined inflamed cyst wall containing polymorphs. H. and E. (× 432.)

Blood-count: Hæmoglobin 78 per cent; W.B.C. 5600; blood-urea 40 mg. per 100 ml. Plain radiographs of the abdomen showed a large mass occupying the right side displacing all the bowels to the left. Intravenous urography showed normal function of both kidneys and normal outlines with no sign of Wilms' tumour. There was no displacement of the ureters. Barium meal showed the stomach, small intestine, and colon to be displaced upwards and to the left, and that there was no bowel at all in the right side of the abdomen (*Fig.* 317). On these findings it was certain that there was a large swelling in the right side of the abdomen, probably arising from the liver.

AT OPERATION (April 20, 1954, T. M.; anæsthetist, Dr. I. M. Gow).—It was obvious, under the relaxation of the anæsthetic, that there was a large, firm swelling occupying the whole of the right side of the abdomen. Through a long right paramedian incision this tumour was exposed. It was of an enormous size and had the shape and appearance of liver. It was carefully delivered and found to be attached to the under surface of the right lobe of the liver, lateral to the gall-bladder, by a bridge of liver tissue which was about 3 cm. thick anteriorly, but

FIG. 316.—*Case* 2. Abdomen distended by mass in liver.

posteriorly gradually became thicker as it fused with the liver generally. Delivery of the tumour caused some deterioration in the child's condition, and an intra-aortic blood transfusion of 5 oz. (150 ml.) was given. The pedicle was then carefully divided after being clamped with large hæmostats and the tumour removed. The large vessels were ligated and the edges of the liver brought together with interrupted catgut sutures. Before the abdomen was closed the patient was given a further 5 oz. (150 ml.) of blood into the aorta. The wound was closed with drainage of the right side of the abdomen.

POST-OPERATIVE PROGRESS.—The child had an entirely uninterrupted convalescence and was discharged home three weeks after operation (*Fig.* 318). She has since been perfectly well and is now in normal health.

THE SPECIMEN.—The excised mass measured 20 × 20 × 10 cm. and weighed 1800 g.—one-sixth of the child's weight. In size and shape it very much simulated a liver. Anteriorly and to the left it came to a thin edge very much as does the normal liver. Posteriorly it was rounded and had in it a large notch where it had been

FIG. 318.—*Case* 2. Abdomen of normal size after removal of mass from liver.

passing over the vertebral column. On section it was seen to consist of liver tissue containing cysts up to 5 cm. in diameter. The larger cysts had a trabeculated lining (*Fig.* 319). Microscopically the cysts were separated by broad bands of cellular fibrous tissue. Next to the cyst cavities was a layer of flat cells. These were not identifiable and could have been either endothelial cells, flattened epithelial cells, or compressed fibrous-tissue cells of the cyst walls. The appearances were thus consistent with both lymphangioma and multiple cysts of bile-duct origin. A decision between these two possibilities is manifestly impracticable; all that can be said is that on grounds of probability a bile-duct origin is the more likely.

The liver tissue surrounding the cystic lesion was congested and the liver cell columns were atrophic. The portal tracts were large and fibrous; each tract contained many large bile-ducts lined by cuboidal epithelium.

FIG. 317.—*Case* 2. Barium radiograph showing stomach and intestines displaced to left.

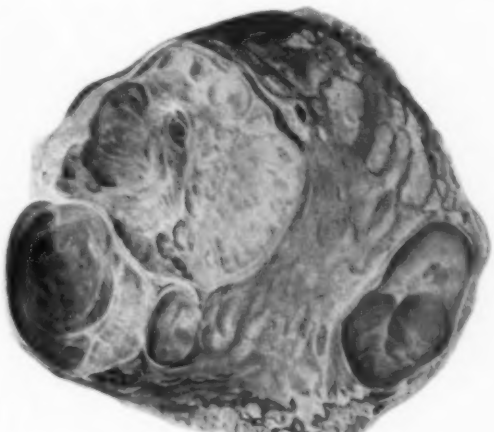

FIG. 319.—*Case* 2. Cut surface of cystic mass from liver.

There was no evidence of malignancy or of active cellular growth in any of the tissues or structures seen in the sections. The conclusion was that the specimen represented a developmental abnormality (hamartoma) of the liver; the changes in the liver substance appeared to be nothing more than pressure effects.

Case 3.—**Polycystic disease of liver and left lung.**

A male aged 67 years was referred to Withington Hospital on June 14, 1949, with severe pain in the left upper abdomen. The pain had awakened him seven days previously at 1 a.m. and had persisted in varying degree; it was made worse by lying down, bending, and by taking deep breaths; he had never experienced this pain previously. For the previous eighteen months he had been easily satisfied by small meals and had lost about one stone in weight. His bowels were regular and there were no urinary symptoms.

On Examination.—He was pale and showed some evidence of weight-loss. His abdomen was prominent, but there was no sign of any free fluid. In the epigastrium,

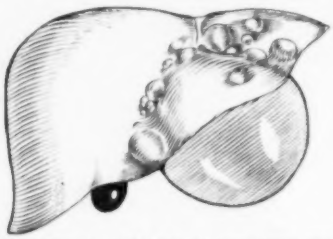

Fig. 320.—*Case* 3. Left lobe of liver shows one large spherical cyst and several small cysts. Small cysts are also present in adjacent part of right lobe. The gall-bladder (black with white spot) is normal.

towards the left side, there was a firm, rounded mass which was slightly tender; its surface was smooth and its margin described as regular. It moved slightly on respiration. There were no enlarged lymph-nodes in the neck or elsewhere and a first tentative diagnosis of carcinoma of the stomach was made. An alternative suggestion was a pancreatic cyst. The patient was admitted the same day and during the next week his abdominal pain steadily diminished; he was taking light diet and there was no vomiting.

The hæmoglobin was 79 per cent, and a barium-meal series showed no sign of any intrinsic lesion in stomach or colon, but the stomach was clearly displaced downwards and to the left by some extragastric mass. The chest radiograph showed no sign of any secondary deposits or cysts in either lung field. On gastric analysis there was a normal resting acid level and no blood was present.

At Operation (A. N.).—On June 23, 1949, the abdomen was explored through a left upper paramedian incision. The palpable mass was found to be a cyst the size of a large grapefruit situated in the left lobe of the liver close to the midline (*Fig.* 320). Small adjacent cysts up to walnut size, but mostly much smaller, were present both in the left lobe and in the adjacent parts of the right lobe. This latter extension to the right felt mainly solid with some cystic areas. Several small hard nodules suggested neoplasm. The remainder of the right lobe of the liver was normal. The alimentary tract, gall-bladder, bile-ducts, pancreas, kidneys, and spleen all appeared normal. The large cyst was aspirated and old altered blood withdrawn. Whether or not the condition was a neoplastic one or a non-malignant cystic condition, the findings suggested that pressure on the stomach was the factor causing symptoms over the past eighteen months, with a recent hæmorrhage into the lesion causing the final acute pain. A resection of the left lobe of the liver was carried out and was greatly facilitated because the main cyst had enlarged to a degree which left a relatively narrow peripheral margin of liver tissue. The extension of the disease into the right lobe was not disturbed.

Post-operative Progress.—Convalescence was uneventful except for the discharge of several ounces of blood-stained fluid from the abdominal drain on the fifth day and some transient diarrhœa during the third week. The patient returned home three and a half weeks after operation. Six months later he had gained 2 st. in weight and he remained well for three and a half years, except that he was never able to take full meals without discomfort and at times complained of slight transient epigastric pain. By June, 1953, he had lost a few pounds in weight and was complaining of slight high epigastric discomfort on swallowing. The right lobe of the liver was palpable, but no recognizable cysts could be felt. Barium meal showed no abnormality. Chest radiographs showed that a number of thin-walled cysts, up to 3 cm. in diameter, were present in the left lung, mainly towards the base. The nature of these 'cysts' was never finally established, but the radiographic appearance was quite compatible with a diagnosis of congenital cysts. During the next twelve months the patient's condition steadily deteriorated with increasing shortness of breath, aggravated by slight exertion and by taking food.

Re-admitted to hospital in August, 1954, he was found to have a large amount of fluid in the left chest, and a total of 110 oz. of slightly turbid blood-stained fluid was removed in three aspirations. The blood was in all probability contamination; no organisms were found and no cells of a malignant type were seen in the fluid. Subsequent chest films again failed to provide a definite diagnosis, but showed, in addition to a considerable residual pleural effusion, an appearance very suggestive of the presence of several giant cysts in the left lung. Returning home, the patient's condition continued to deteriorate, with increasing dyspnœa, and he died in December, 1954. There was no autopsy to establish the full pathology in the lung, but the clinical and radiological evidence supports the view that death was due to a progressive cystic condition in the lung, which might well be related in aetiology to the benign cystic condition in the liver.

The Specimen (Dr. Lois Stent).—"The specimen consists of a portion of a left lobe of the liver which contains a cyst approximately 14 cm. in diameter which has been opened. The surface is smooth and deeply congested and the lining traversed by broad fibrous bands. At one point on the surface of this large cyst are two smaller cysts 2 cm. in diameter; the surfaces are smooth and the fluid aspirated is clear and colourless." Microscopically: "The lining of the smaller cysts was smooth and traversed by fibrous bands. Histologically the large cyst is lined by flattened epithelium and the wall consists of fibrous tissue in which are embedded numerous small bile-ducts. Near the lining the fibrous tissue contains large cells filled with blood-pigment. The liver tissue shows widened portal tracts containing dilated bile-ducts, and separation of liver parenchyma by groups of small bile-ducts surrounded by a vascular stroma containing lymphocytes and eosinophil leucocytes. Some of these areas of budding bile-ducts are infiltrated with leucocytes and the sinuses in the surrounding liver tissue are dilated and filled with leucocytes. There is no evidence of neoplastic change and the appearances are those of multiple cystic disease of the liver with a portal cirrhosis. The fluid aspirated from the cysts contained urea 85 mg./100 ml. and cholesterol 125 mg./100 ml. No bile was present."

Case 4.—**Polycystic disease of liver.**

A female aged 65 years. Three weeks prior to admission to the Christie Hospital in May, 1956, she had an attack of diarrhœa and, rubbing her abdomen, which felt uneasy, she found a hard mass on the right side. She was small, of spare build, and very active. Her general health had been good though she had had a number of surgical operations—1927, removal of left ovarian cyst (non-malignant); 1929, hæmorrhoids and fistula; 1931, tonsillectomy; 1937, hysterectomy for fibroids. She had recently lost a few pounds in weight. Her appetite and digestion were good. The bowels had been constipated for many

years. The patient was emphatic that she had had no previous abdominal discomfort of any sort, and that she had been completely unaware of anything wrong in the abdomen until the swelling was felt quite accidentally.

On Examination.—The patient, being of spare build, was easy to examine. There was a mass present extending from under the right costal margin; it was rounded, smooth, and firm, and moved on respiration. It could also be moved from side to side and movement could be transmitted through from the right loin. A note was made "very suggestive of large right kidney but could be gall-bladder". Rectal examination was negative.

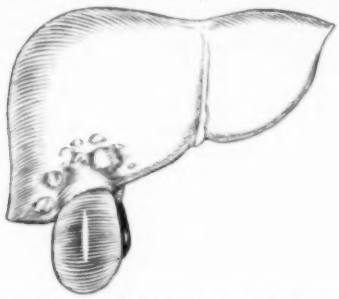

Fig. 321.—*Case* 4. Right lobe of liver shows one large elongated cyst and several small cysts above it. Part of the gall-bladder (black with white streak) is visible at the left edge of the largest cyst.

The hæmoglobin was 96 per cent. Renal investigations, including cystoscopy and ascending pyelogram, showed no sign of any pathology in the renal tract. Chest radiograph was normal. Cholecystogram was reported: "No gall-stones are identified. Graham's test shows what appears to be a gall-bladder constricted or compressed to form a proximal dilated portion and a moderately contracted fundal portion." Barium-meal X-ray examination: "Stomach small, mobile, and normal. Duodenum and jejunum also normal. Meal has passed on normally to terminal ileum and colon at 6 hours. Normal but slow onward passage to 24–30 hours. No organic disease in alimentary tract is revealed. Liver is either low or enlarged and seems to displace hepatic flexure down towards right iliac fossa. Right-sided mass thus shown to be of either hepatic or gall-bladder origin."

At Operation (A. N.).—On June 2, 1956, the abdomen was explored through a right upper paramedian incision. The palpable swelling was a large thin-walled cyst in the right lobe of the liver, growing from the free margin; it was elongated and 5–6 in. in the longest axis (*Fig.* 321). The fundus could be delivered into the wound. Deep to this, and with only a small part of its wall showing, was another smaller cyst. There were a number of much smaller adjacent cysts up to 2 cm. in size. The gall-bladder was pushed down and back by the cysts, but otherwise appeared normal. Stomach, duodenum, pancreas, spleen, and kidneys felt normal. Apart from the cysts the liver tissue looked normal. There were many adhesions in the lower abdomen. Five-sixths of the projecting cyst was excised and hæmostasis secured by No. 1 catgut sutures through the cyst wall and adjacent liver tissue. The small residual area of cyst wall was diathermized. No attempt was made to excise any of the deeper cysts. The chief deep cyst was opened, emptied, and drained. The fluid from both cysts was slightly turbid. The cyst lining was smooth and nothing was seen or felt to suggest other than simple cysts, presumably of congenital origin.

Post-operative Progress.—Convalescence was uneventful. The drain into the deeper cyst was left in situ for ten days and on two occasions 10 ml. of lipiodol was injected down the drain into the cyst cavity to discourage further secretion from the cyst lining. When seen three months after operation the patient was well and free from symptoms. The liver was not palpable.

The Specimen (Dr. Helen M. Russell).—"Liver tissue showing the wall of a large cyst and also smaller cysts. The liver parenchyma itself shows irregularly developed cells, and fibrosis and cell infiltration of the portal tracts, all of which may be merely the effects of pressure and disturbed circulation. There is one histological tubercle (miliary size) in four sections examined. Report on the fluid obtained from the cysts: Fouchet's test for bile-pigment was negative. Microscopic examination: a few red cells and mononuclear cells. No cysts or scolices present. The absence of bile suggests that these are congenital cysts. Culture—no growth obtained."

DISCUSSION

Diagnosis.—When evident clinically, a cystic liver usually presents as an abdominal swelling. This may be difficult to localize and may suggest any space-occupying lesion of the liver, gall-bladder, kidney, pancreas, ovary, omentum, or mesentery. The swelling may be the only complaint or may be accompanied by symptoms due to pressure of the mass on neighbouring viscera; chronic dyspepsia may result from displacement of the stomach or duodenum, and obstructive jaundice from compression of the common bile-duct; constipation or diarrhœa may follow distortion of the transverse colon, and ascites and œdema of the legs may reveal portal obstruction; palpitation, dyspnœa, and cyanosis may be caused by elevation of the diaphragm.

Rarely, a large cyst may present as an acute abdominal emergency owing to torsion of the pedicle (Orr and Thurston, 1927), intracystic hæmorrhage (Ackman and Rhea, 1931; our *Case* 3), or intraperitoneal rupture (Horton, 1954). Hæmorrhage into a cyst is signalled by sudden severe pain, but discharge of cyst contents into the peritoneal cavity causes little pain or irritation. The cysts may be numerous enough to cause hepatic insufficiency—two examples were reported by Niemetz, Sokol, and Meister (1949)—the insufficiency may result in death, as in a case mentioned by Taft (1952) and a remarkable case reported by Deutsch (1953); in this case, hæmorrhage occurred into many cysts which became converted into abscesses, the patient dying in hepatic coma. Carcinoma of the cysts in cystic livers has been reported by Willis (1943) and Richmond (1956).

Diagnosis of cystic liver can be satisfactorily made only by laparotomy, which has the further advantage of permitting immediate treatment; less radical methods are inconclusive, waste time, and needlessly inconvenience the patient. Punch biopsy of the liver may show that the lesion is cystic, as it did in the cases of Caravati, Watts, Hopkins, and Kelly (1950) and Taft (1952), but will not finally settle its nature, and might disseminate infection or tumour. Niemetz and others (1949) found aspiration biopsy inadequate and misleading and diagnosed their two cases by peritoneoscopy, although the histories would have warranted immediate laparotomy. Caravati and others (1950) thought that intravenous thorotrast might demonstrate a large smooth filling defect.

Treatment.—It is best to resect the cyst, or the affected part of the liver if the cysts are multiple.

The whole left lobe has been successfully removed in cystic disease; this was done for a solitary cyst by von Haberer (quoted by Jones, 1923), for multiple cysts by Clagett and Hawkins (1946) and one of us (A. N., *Case* 3), and for carcinoma of cysts in the case reported by Richmond (1956). Clagett and Hawkins thought resection justifiable if multiple cysts were causing discomfort, provided that the kidneys were normal on palpation; they thought that nothing should be done if the kidneys were seriously involved, or if there were no symptoms referable to the liver.

The operation itself is, of course, not new; left hepatic lobectomy after ligation of artery, portal vein, and hepatic duct has been reported on a number of occasions (Jackson, 1931; Abel, 1934) and may be regarded as a formal procedure. Access and hæmorrhage are the problems confronting surgeons undertaking removal of hepatic tumours and cysts. The site of the lesion will determine the choice between abdominal, thoracic, and abdomino-thoracic approaches. Operative hæmorrhage may be difficult to control, but secondary hæmorrhage should be a thing of the past with modern antibiotics. Liver tissue may be sectioned with knife or diathermy, the assistant using fingers or light clamps to give temporary control of bleeding. Compression of the hepatic artery and portal vein between the assistant's finger and thumb below the liver may help. More recently, Ogilvie (1953) has described a method of dissection using the closed points of a fine hæmostat which promises to facilitate these operations. Large vessels can be caught and tied, general oozing being arrested by catgut mattress sutures and gelfoam. As regards complications, biliary fistula is unlikely to persist if the main bile-ducts are patent.

Cysts that could not be removed have been marsupialized and often irrigated with protein coagulants or other substances capable of destroying the cyst lining; Zenker's solution was successful in the cases of Montgomery (1940) and Munroe (1942); Carnoy's fluid was found effective by Davis (1939), and quinine-urethane and lithocaine by Maingot (1940). Hepatic cysts have also been treated by anastomosis to the duodenum (Burghard, reported by Everidge, 1914) or stomach (Manheimer, 1953)—methods now familiar in the treatment of cysts of the pancreas (Sinclair, 1955). Manheimer concluded that in his patient the cyst became obliterated, most probably by the scarifying action of the hydrochloric acid of the gastric juice. It is uncertain what really happened; there was no radiographic evidence of the cyst fourteen months after operation, but since barium rarely enters pancreatic cysts joined to the stomach, it may well have failed to enter the hepatic cyst similarly treated in this case.

Pathology.—Hepatic cysts could in theory take origin from a variety of hollow structures in or near the liver. The most obvious potential sources of cysts are bile-ducts, and various suggestions have been made as to how cysts might be formed from these. Moschcowitz (1906) concluded that bile-ducts giving rise to cysts are aberrant, but Rolleston (quoted by Boyd, 1913) thought that the structures regarded by Moschcowitz as aberrant bile-ducts were the microscopic cysts of a cystic liver. A different mechanism was suggested by Norris and Tyson (1947), who concluded that bile-ducts yielding cysts are normal, but of transitory type; they observed distortion, segmentation, and dilatation, interpreting these changes as an abnormal extension of the normal process of resorption in the first few generations of bile-ducts. Yet another opinion is that bile-duct cysts are cavernous biliary angiomas.

Other possible sources of hepatic cysts are lymphatic channels, mucous glands in the biliary passages (Davis, 1937), and the ligamentum teres (Wakeley and MacMyn, 1931). These authors thought that solitary cysts of the liver might arise from failure of closure of the fœtal left umbilical vein at the hepatic end, and that a cyst so formed could acquire a false capsule of compressed liver tissue; this possibility was suggested by their *Case* 3 (a cyst of the ligamentum teres), and in support of it they mentioned the common occurrence of solitary hepatic cysts near the insertion of the ligamentum teres and of cysts in the adult remnants of other fœtal structures.

While it is easy to suggest theoretically possible origins for liver cysts, it is often impracticable to identify the source of the cysts in individual cases; this is so especially when the cyst is lined by flat cells or without lining. In an epithelial organ, the presumption must always be that any cyst is of epithelial origin, even when the characteristic epithelium is not recognizable owing to flattening, destruction, or loss through desquamation. In the liver, therefore, it is most reasonable to assume a bile-duct origin, however little the wall of the cyst may resemble the wall of a bile-duct. Flattened bile-duct epithelium might simulate lymphatic endothelium and so lead to the diagnosis of lymphangioma. Some hepatic cysts may well be genuine lymphangiomas, but the diagnosis would be difficult to establish.

A cyst once formed, no matter what its origin, could conceivably have various types of life-history as the result of one or more of the following physical processes: (1) Enlargement; (2) loculation; (3) coalescence of loculi; (4) conglomeration with other cysts; and (5) coalescence with other cysts. Three main types of life-history can be suggested:

a. Simple Enlargement.—Starting as a microscopic cavity the cyst could enlarge without loculation as fluid accumulated; ultimately it could reach macroscopic dimensions, and in rare cases it could form a clinically palpable mass.

b. Enlargement with Loculation.—A microscopic cyst could enlarge up to a certain point and then become loculated. If fluid continued to accumulate within the loculi, their walls might undergo pressure atrophy, with consequent coalescence of the loculi and the ultimate formation of a unilocular cyst. A single cyst could thus pass through three stages: unilocular, multilocular, and once again unilocular.

c. Enlargement and Conglomeration with Other Cysts.—The cyst could enlarge until it came in contact with other cysts; together with these it could form a conglomeration that could be described as a multilocular cyst. The walls of the independently formed cysts forming the conglomeration might undergo pressure atrophy, the resulting structure being a variety of unilocular cyst.

A *multilocular* cyst could thus be either (i) a single cyst that had become loculated, or (ii) a conglomeration of independently formed cysts.

A *unilocular* cyst could be either (i) a single cyst that had never become loculated; (ii) a single cyst that had become loculated and then lost its loculi; or (iii) a cavity resulting from loss of partitions in a conglomeration of independently formed cysts. The trabeculated lining found in many unilocular cysts may represent the walls of cavities that have coalesced, as suggested by Davis (1937).

Hepatic cysts have long been classified on a purely numerical basis as multiple and solitary. When the surgeon finds a number of cysts well separated from each other the diagnosis will obviously be multiple cysts (polycystic disease); the only problem —insoluble though it may be—is where the cysts arose: in bile-ducts, lymphatics, or some other structures. When only one cyst is found (solitary cyst), the problem of interpretation is much more difficult, especially as a solitary cyst may be related to polycystic disease in various ways, or even be a form of this disease.

Four possible interpretations of a solitary cyst may be considered:—

1. The solitary cyst may be demonstrably of traumatic or parasitic origin and the question of a relation to polycystic disease does not arise.

2. The solitary cyst may have no obvious cause; in this case it may in fact be the only cyst found in a polycystic liver, other cysts being missed because inaccessible or very small. To exclude the presence of such cysts, pathological examination of the entire liver would be needed; this will very rarely be feasible since patients normally survive operations for the relief of cystic disease.

3. The solitary cyst may be a conglomeration of independently formed cysts, or a single cavity formed by the coalescence of such cysts; in both these cases the solitary cyst is really a localized form of polycystic disease. The pathologist is unlikely to be able to differentiate between (*a*) a solitary cyst formed by conglomeration of multiple cysts and (*b*) a solitary cyst derived from a single original cavity.

4. The solitary cyst may be derived from a single cavity of the same nature (for example bile-duct or lymphatic) as the multiple cysts of polycystic disease; in this case the only difference between the solitary cyst and multiple cysts is one of number.

In brief, the positive diagnosis of polycystic disease is easily established; the difficulty is to exclude this disease when only one cyst (solitary cyst) is found. In practice, polycystic disease will normally be excluded only when the solitary cyst is clearly of traumatic or parasitic origin.

SUMMARY

Four cases of cystic disease of the liver are described: (1) A cystic hamartoma in a boy aged 3½ years; (2) a cystic hamartoma in a girl aged 10 months; (3) polycystic disease of the liver and left lung in a man aged 67 years; and (4) polycystic disease in a woman aged 65 years. In each case the liver lesion was excised, wholly or partly.

A cystic liver usually presents as an abdominal swelling, best identified by laparotomy. The ideal treatment is removal of the lesion, the site of the lesion determining the choice between abdominal, thoracic, and abdomino-thoracic approaches. Hepatic cysts have also been treated by marsupialization or internal drainage.

Hepatic cysts could originate in bile-ducts, lymphatics, mucous glands, or the ligamentum teres. Whatever its origin, a cyst could simply enlarge, or enlarge with loculation; it could also form a conglomeration with other cysts. Breakdown of loculi in a single cyst, or of the cysts forming a conglomeration, could result in a unilocular cyst, perhaps with a trabeculated lining.

When only one cyst is found (solitary cyst), it may be difficult to exclude the presence of polycystic disease. A solitary cyst may, in fact, be the only cyst found in a polycystic liver; if formed by conglomeration of multiple cysts, a solitary cyst is itself a localized form of polycystic disease; if derived from a single cavity, a solitary cyst may be of the same nature as the multiple cysts of polycystic disease. In practice, polycystic disease can normally be excluded only if the solitary cyst is clearly of traumatic or parasitic origin.

Figs. 313–315 are by Mr. N. Mowat, *Fig.* 317 is by the Department of Medical Illustration, Manchester Royal Infirmary, and *Figs.* 319–321 are by Miss D. Davison.

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AN ORAL APPROACH TO PAROTID TUMOURS WITH PHARYNGEAL EXTENSION

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MIXED salivary tumours may arise not only in the three named major salivary glands, but also in minor salivary glands of the lips, cheeks, palate, floor of mouth, nasal cavity, and pharynx. Of these sites, it is recognized that more arise in the palate than in any other structure of the head and neck except the parotid gland (Martin, 1942). Parotid gland in size "tended to push outward between the mastoid and the ascending ramus of the mandible", to appear externally "as a mass close up under the auricle", which is regarded as "an extension from the pharyngeal tumour".

Such a view is no longer tenable following the careful researches of Patey and Thackray (1957)

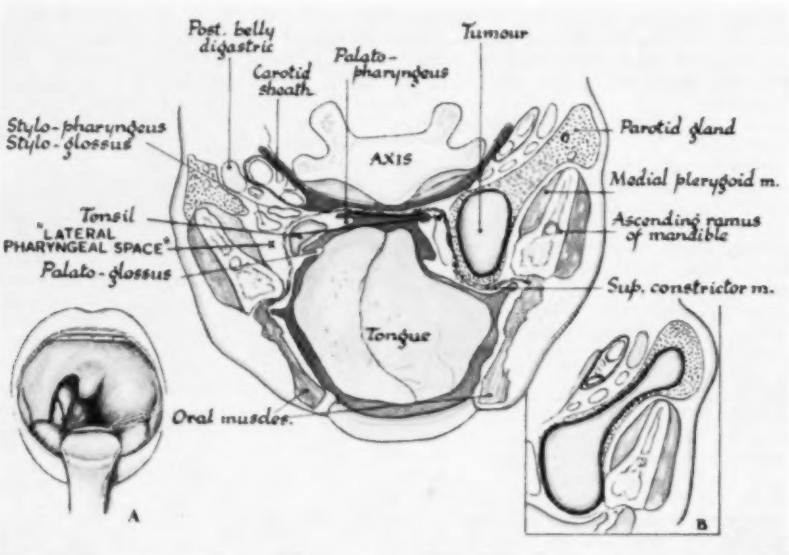

FIG. 322.—Transverse section through the head at the level of the axis vertebra and passing through the oral cavity. The right side is drawn from an actual specimen; the left side represents a diagrammatic reconstruction to show the invasion of the lateral pharyngeal space by a tumour arising in the deepest part of the parotid gland. *Inset* A, Drawing of intra-oral appearances, resulting from the presence of a left parotid tumour with pharyngeal extension. *Inset* B, Diagrammatic reconstruction of a tumour of the deep lobe of the parotid gland, of the dumb-bell variety.

predominance is, however, such that only 5 mixed salivary tumours are palatal for every 100 found in the parotid (Willis, 1953).

It has long been known that tumours arising primarily in the parotid gland may rarely produce a swelling in the lateral pharynx. Thus Stein and Geschickter (1934) found that of 113 cases of tumours of the parotid gland 7 (5·2 per cent) presented in the 'throat', the presenting symptom in 2 patients being dysphagia.

Despite this, even recent publications have discounted the parotid gland as a source of mixed salivary tumours of the soft palate and faucial region.

Thus, in a recent report (Havens and Butler, 1955) of 41 mixed salivary tumours of the pharynx, of which 7 also presented as external swellings in the parotid region, the belief is recorded that the latter were of primarily pharyngeal origin, but with increase and Patey and Ranger (1957) into the surgical and pathological anatomy of the parotid gland.

The parotid gland is ensheathed by the investing layer of cervical fascia, which provides a dense fibrous capsule on the superficial surface of the gland, but merely a tenuous areolar covering on its deep aspect. A tumour of the deep portion of the parotid (i.e., that part of the gland deep to the facial nerve—the 'retromandibular lobe' of American writers) therefore tends to grow in the direction of least resistance, deeply into the parapharyngeal region by traversing the stylomandibular tunnel, an opening bounded above by the base of the skull, anteriorly by the ascending ramus of the mandible and medial pterygoid muscle, and posteriorly by the styloid process and stylomandibular ligament. Having thereby reached the lateral or parapharyngeal space whose lateral wall (the ascending ramus of the mandible covered by the medial pterygoid muscle) and posterior wall (the carotid sheath attached to the base of skull above, and lying on the prevertebral muscles

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and transverse processes of the upper cervical vertebræ), are relatively inextensible, subsequent enlargement of the tumour takes place at the expense of the medial wall, constituted at this level by the superior pharyngeal constrictor.

As a result, on looking inside the mouth a characteristic bulging of the lateral soft palate is seen, with medial displacement of the tonsil and pillars of the fauces of the corresponding side, the displacement sometimes bringing the uvula into contact with the unaffected side (*Fig.* 322, *inset* A). The overlying mucosa is not attached to the underlying growth, and though distended, remains free of ulceration. The tumour thus tends to assume a dumb-bell shape, with the zone of constriction between the two globular extremities situated at the site of the stylomandibular tunnel (*Fig.* 322, *inset* B). On bi-digital examination of the internal and external swellings, ballottement can usually be obtained between the internal and external examining fingers, and constitutes an almost diagnostic sign.

With further enlargement the growth may cause pressure atrophy of the styloid process and stretching of the stylomandibular ligament; the resistance of the walls of the lateral pharyngeal space then results in moulding of the tumour into a large spherical or ovoid mass, entirely without its early dumb-bell configuration (*Fig.* 322). Subsequent enlargement may then occur inferiorly as well as medially, the lower pole of the tumour passing downwards medial to the ascending mandibular ramus to present as a subcutaneous swelling in the region of the angle of the jaw, or the posterior part of the digastric triangle. Thus in 2 of the cases reported below, and in all 12 of the cases reported by Morfit (1955), there was no dumb-bell contour to be remarked in the enucleated tumours, nor was there any external swelling present, except occasionally below the level of the horizontal ramus of the mandible in the largest growths.

Since a tumour able to enter the lateral pharyngeal space must of necessity arise in that small plug of parotid gland blocking the stylomandibular tunnel, it is understandable that such pharyngeal extensions must be of rare occurrence. Thus Patey and Thackray (1957, 1958) found that of 80 parotid tumours, only 4 showed such a development.

All the tumours are large. In Patey's cases the longest diameter varied from 3·75 to 7·5 cm., and in the series reported below, from 4 to 7 cm. The explanation of this is two-fold. First, the rate of growth of mixed tumours is very slow; McFarland (1936) estimated that it took ten years to reach the size of a walnut, and twenty years to become as large as an orange. Secondly, since most or all of the tumour presents as a faucial or pharyngeal swelling, the growth is for much of its life-history a silent one, and produces late symptoms only.

MATERIAL

Four patients having pharyngeal extensions from primary mixed tumours of parotid origin have been treated in this unit during the period 1948–58.

All cases showed the characteristic intra-oral appearances already described (*Fig.* 322 A), and all except one also presented a palpable external swelling, which rendered ballottement possible on bi-digital palpation.

The sex distribution was equal, and ages varied from 40 to 73 years.

In all cases the tumours were large and underwent surgical enucleation through the mouth.

Only one growth has so far recurred; this was in the case of the first dumb-bell tumour encountered, which ruptured during enucleation.

A case of fibroma is also described.

OPERATION

The tumour is enucleated through an oral incision, made at the site shown in *Fig.* 323.

This approach, devised by Hynes (1951), is based upon the incision employed in the classical operation of V–Y retroposition of palatal mucosa used in the operative repair of congenital clefts of the palate.

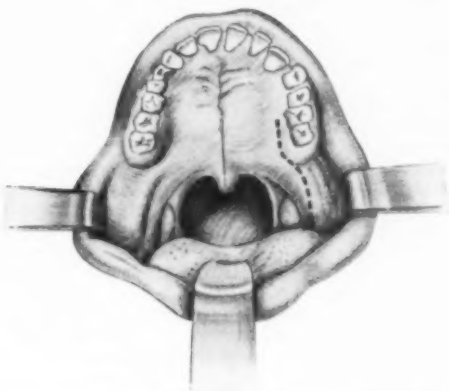

FIG. 323.—Semi-diagrammatic, showing site of incision used in oral approach to the lateral pharyngeal space.

In that operation the incision in the lateral part of the hard palate is extended posteriorly behind the maxillary tuberosity in order to expose the hamular process of the medial pterygoid plate, which is then fractured. At this stage of cleft-palate operations the glistening tendinous fibres of origin of the medial pterygoid muscle can be observed in the lateral part of the wound, at the site of their attachment to the medial aspect of the lateral pterygoid plate; medially, the hamulus of the medial pterygoid plate is crossed by the shining tendon of the tensor palati muscle, and provides the upper bony origin of the superior constrictor muscle of the pharynx. In the fissure between the two pterygoid plates lies the plane between the medial pterygoid muscle and the superior pharyngeal constrictor, which constitutes the anterior boundary of the lateral pharyngeal space.

The patient is anæsthetized through a cuffed intratracheal tube passed through the nose, and the mouth is held widely open by a Dott's gag.

The incision, which deviates only a little from being a straight line, begins on the hard palate near the gingival margin at a point about 1½ cm. anterior to the maxillary tuberosity, passes backwards parallel to the maxillary molar teeth, and curves behind the tuberosity of the maxilla before descending along the line of the pterygomandibular ligament to its

termination; it is usually about 5 cm. long, but this is varied according to the size of the tumour.

The incision is deepened through the stretched muscle overlying the tumour, until the capsule enclosing the latter is exposed. Enucleation of the growth is then completed by blunt dissection, at first using curved dissecting scissors, but later by digital dissection, the finger being swept round the tumour as it lies in the lateral pharyngeal space in the plane between the superior constrictor and the medial pterygoid muscles. Enucleation is accomplished in much the same manner as during a Freyer's prostatectomy.

External pressure exerted over the region of the lower pole of the parotid gland by the assistant aids materially in elevating the tumour within the arc swept by the enucleating finger.

By this means, and with patience, very large tumours can be delivered safely into the mouth and removed.

As the tumour lies deep to the facial nerve, the latter is never at any time placed in hazard.

The wound is loosely sutured in its upper part, but left open below to provide natural drainage.

In no case has a pack to control hæmorrhage been required, to be introduced into the cavity left after evacuation of the tumour, nor has the external carotid artery been ligated in any patient.

All patients have maintained post-operative nutrition by natural mouth-feeding; a fluid diet is usually used for 3–5 days, and a soft-solid diet for a further 10–14 days.

In all cases the muscular movements of soft palate and jaw have been unimpaired, and there has been no oral or pharyngeal distortion from contracture.

All patients were discharged from hospital 10–21 days after operation.

The first dumb-bell tumour encountered ruptured during delivery, resulting in repeated recurrence of growth. The second such tumour was therefore dealt with by making an additional external incision, enabling it to be removed successfully in two halves through this combined interno-external approach.

CASE REPORTS

Case 1.—Male, aged 64 years, with a history of difficulty in swallowing due to swelling in the throat for one and a half years. No pain. An attempt made elsewhere one year previously to remove the tumour through a pharyngeal incision had failed. Deep X-ray therapy had been given without effect.

On Examination.—A large, solid, intra-oral swelling was found to be displacing the left tonsil and soft palate downwards and medially; it was adherent to the pharyngeal wall at the site of previous incision. No external swelling was noted.

At Operation.—Enucleation through the oral approach as described above. The tumour was encapsulated, spherical, and of 4 cm. diameter.

Progress was uneventful, except for pain in the left ear and a slight hæmorrhage from the operation wound on the fifteenth post-operative day. Both subsided without active intervention.

Pathology showed a mixed tumour of salivary gland.

Progress.—The patient died three years after operation of coronary thrombosis. There was no evidence of recurrence of growth at any time.

Case 2.—Male, aged 47 years, with a history of slight but increasing deafness in the left ear for four years, together with swelling of the left parotid gland and left side of the throat. Deep X-ray therapy had failed to arrest growth.

On Examination.—A swelling 4 × 2 cm. in size at the lower pole of the left parotid gland; intra-orally a marked bulge of the left soft palate and tonsillar region displaced the uvula to the right. Ballottement was obtained.

At Operation.—Attempted enucleation through the described oral approach failed because of dumb-bell extension of the growth. The capsule ruptured, and 120 g. of tumour were removed piece-meal.

Post-operative recovery was uncomplicated. Pathology showed a mixed salivary tumour.

Progress.—The tumour recurred four years after operation. Five subsequent operations for recurrent growths have been completed. On the last occasion a wide external approach to the parotid region was made, and the facial nerve and its branches dissected out; this was achieved without difficulty, for the superficial parotid gland enclosing the nerve was essentially normal. Despite osteotomy of the ascending ramus of the mandible to improve access, inoperable extension of growth to the base of the skull prevented its complete removal.

Following conventional deep X-ray therapy in heavy dosage, the patient continues to work and appears well, albeit with palpable and inoperable recurrence of growth ten years after the initial operation.

Case 3.—Female, aged 73 years, with a history of swelling in the left submandibular region below the angle of the jaw for ten years. This caused no symptoms and was not enlarging. Swelling in the left side of the throat for 6 months had caused no symptoms, but was increasing in size.

On Examination.—There was a palpable swelling about 3 cm. in diameter, at the lower pole of the left parotid gland. Intra-orally, a marked bulging of the soft palate and faucial region on the left side resulted in displacement of the uvula to lie in contact with the right faucial pillars. Ballottement was obtained.

At Operation.—Through an intra-oral incision the pharyngeal extension of the tumour was mobilized, but its dumb-bell shape prevented enucleation from being completed. Using an external incision superficial conservative parotidectomy was performed, the branches of the facial nerve retracted, and mobilization of the superficial lobe of the tumour achieved.

Following division of the attenuated isthmus joining the two lobes of the tumour, the latter was removed, the superficial lobe (measuring 5 × 4 × 2·5 cm.) through the external wound, and the deep lobe (measuring 6·5 × 3·5 × 2·5 cm.) through the mouth. The external incision was closed with drainage, and the intra-oral incision carefully sutured.

Post-operative progress was uneventful.

Pathology showed a mixed salivary tumour.

Progress.—Satisfactory progress maintained one year after operation.

Case 4.—Female, aged 40 years, with a history of swelling of the left parotid region for twelve years. About nine months previously difficulty in swallowing had drawn attention to a swelling of the left tonsillar region. During recent months attacks of pain had occurred over the left side of the head. Six months previously deep X-ray therapy had been given without response.

On Examination.—An external swelling about 2·5 cm. in diameter was palpable just behind and below the left angle of the mandible.

The internal swelling presented as a characteristic bulge of the left soft palate and tonsillar region, considerably displacing the uvula. Ballottement was obtained.

At Operation.—Employing an intra-oral approach a large encapsulated tumour measuring 6 × 6 × 3 cm. (*Fig.* 324) was enucleated.

The patient made an uninterrupted recovery.

Pathology showed a mixed salivary tumour.

PROGRESS.—No evidence of recurrence, and pain over the left cranium has ceased, three months after operation.

FIG. 324.—*Case* 4. Tumour measuring 6 × 6 × 3 cm., of mixed salivary type.

Case 5.—Female, aged 48 years. This patient had received sanatorium treatment for pulmonary tuberculosis at the age of 26 years, and was apparently cured.

Two years ago a swelling appeared in the left submandibular region, and on routine examination at the chest clinic a swelling was also noted in the left faucial region, displacing the left tonsil.

ON EXAMINATION.—A firm ill-defined external swelling was present just below the left mandibular angle, and a characteristic bulging of the left soft palate and tonsillar region was found inside the mouth. Ballottement was obtained. A diagnosis of tumour of parotid origin was made.

AT OPERATION.—Through an intra-oral incision an encapsulated, lobulated tumour measuring 7 × 4 × 4 cm. was enucleated; delivery of the growth into the mouth was only completed after the coronoid process had been removed and the ascending ramus of the mandible divided. Jaw immobilization by suitable intra-oral splints was subsequently maintained for six weeks, after which normal jaw function was restored within a few days.

PATHOLOGY.—The growth proved to be composed entirely of dense hyaline connective tissue. There was no evidence of healed tuberculoma. The lesion probably represented a massive fibroma, which had undergone extensive hyaline degeneration.

PROGRESS.—The patient continues to be well, and free from recurrence five years after operation.

DISCUSSION

Symptomatology.—The tumour being of slow growth, and anatomically situated so as to involve essential structures only at an advanced stage of development, symptoms appear late.

Thus, in the cases reported above, swelling or symptoms had been present for periods of from 9 months to 12 years, but in reality the tumour must have been present in every case for much longer periods. Morfit (1955) records that in one case the growth had been present for 30 years.

In the present series, slight dysphagia was the presenting symptom in 2 of the 5 cases, slight but increasing deafness on the affected side in another (presumably due to local occlusion of the pharyngotympanic tube by growth), and a swelling in the throat was incidentally noticed in the last two cases. One patient developed pain over the distribution of the auriculotemporal nerve, presumably from direct pressure, since removal of the tumour cured the pain.

Previously reported cases have presented with vague complaints of 'something sticking in the throat', or a 'feeling of pressure', or 'fulness' (Morfit, 1955), and the intra-oral swelling has often been misdiagnosed as a peritonsillar abscess (Havens and Butler, 1955; Patey and Thackray, 1957), or merely discovered because the upper denture no longer fitted (Montreuil, 1958).

Pain is rare, and in no case has facial nerve involvement by growth occurred.

Differential Diagnosis.—It has been long established that the exact nature of parotid swellings cannot be ascertained on clinical grounds alone.

It is also recognized by most authorities that preoperative biopsy is contra-indicated because of danger to the facial nerve (Redon, 1953), and the possibility of disseminating tumour cells beyond the limits of the capsule (Martin, 1942; Patey and Thackray, 1958).

The difficulties of clinical diagnosis are well illustrated in *Case* 5, which was a fibroma, and was enucleated by the same method as the cases of mixed salivary tumours.

Although fibromata may occur in the parotid gland (Aird, 1957) they are very rare. Similar tumours to that described above have been reported as 'latero-pharyngeal fibroma' by continental writers (Vaheri and Harma, 1957), and Scalori (1954) found 20 such cases recorded in the literature. In view of the anatomical situation of the tumour and the physical signs present, it is suggested that a parotid origin for these unusual growths must be considered.

Other tumours having a clinical appearance indistinguishable from mixed salivary tumours presenting in the lateral pharynx are fibrolipoma (Howarth, 1930), lymphoma (New and Childrey, 1931), neurinoma, lymphatic leukæmia limited to the parotid lymph-nodes (Patey and Thackray, 1957), and neurofibroma (Cranmer, 1958). Aneurysm of the internal carotid artery (which, however, shows expansile pulsation) and carotid body tumour (which presents largely in the neck) can also cause bulging of the lateral pharyngeal wall (Morfit, 1955). Other lesions meriting consideration are chordoma and cold abscess.

Patey and Thackray (1957) point out that carcinoma of the parotid gland may spread to the parapharyngeal region, but at such an advanced stage cranial nerve involvement is likely, and the clinical picture is quite different, since carcinoma does not observe anatomical boundaries.

Treatment.—The treatment by enucleation of parotid mixed tumours presenting externally only underwent modification when it was realized that the method was followed by a tumour recurrence rate of about 25 per cent (McFarland, 1936, 1943).

As a result, following the pioneer work of Janes (1940), Bailey (1941), and Redon (1953) the principle of conservative parotidectomy (whereby excision of

the growth, together with a variable amount of surrounding normal parotid tissue, was achieved with conservation of the facial nerve and its branches) has become widely and currently accepted practice (Patey, 1954). Inevitably, by such a procedure the likelihood of surgical injury to the facial nerve was increased, but the incidence of growth recurrence has been greatly diminished, in the case of primary mixed tumour to such levels as 9 per cent (McCune, 1951), zero (Redon, 1953), and 6·5 per cent (Janes, 1957).

On the other hand, the treatment by enucleation and delivery through the mouth, of that variety of parotid tumour which produces a pharyngeal extension, has continued to be used up to the present time. The incision has usually been made in the pharynx over the most prominent part of the tumour, though Ehrlich (1950) used an oral incision made through the palatopharyngeal fold, but found ligation of the external carotid artery necessary to control bleeding.

Criticisms of the oral approach, that it would not permit the removal of the larger tumours (Morfit, 1955), and that the dangers of operative hæmorrhage and post-operative pharyngeal contracture would be excessive (Beales, 1955), are without foundation in fact when the operative approach described above is employed.

A tendency has become manifest in recent years, however, to bring the surgery of parotid tumours with pharyngeal extension into line with the treatment of parotid growths in general; that is, by wide external exposure followed by total conservative parotidectomy (Patey and Thackray, 1957). It is evident, however, that this method, though described as an 'excision' of growth, is, in fact, not rarely achieved by digital enucleation in the late stages of the operation (Martin, 1957).

As a compromise measure, Morfit (1955) has maintained the cause of enucleation following a wide submandibular external incision.

In the case of these tumours, the decision between an internal and external surgical approach must be chiefly determined by consideration of two factors: (1) The incidence of post-operative facial nerve paralysis, and (2) growth recurrence rate.

1. *Post-operative Facial Nerve Paralysis.*—Such a post-operative complication is relatively the more serious, because mixed parotid tumours have a good prognosis as regards life expectancy; only 2 per cent of sufferers die of their tumour (Aird, 1957).

In the context of total parotidectomy for primary mixed tumour (which would be the operation usually required in cases of parotid tumour with pharyngeal extension, if an external approach were used), McCune (1951) found that 9 per cent of cases sustained a permanent partial facial paralysis.

Marshall and Miles (1947), operating on primary mixed tumour at all sites, and dissecting out the facial nerve before excising the growth, found that partial facial nerve paralysis followed in 5·4 per cent and total paralysis in 2·7 per cent. It would seem reasonable to assume that these figures may be increased if all the growths considered were deep to the facial nerve.

2. *Recurrence Rate.*—The recurrence rate following enucleation of parapharyngeal mixed parotid tumours may justifiably be estimated as falling appreciably below the estimate of 25 per cent (which is based upon an analysis of mixed tumours of all sizes and at all sites), because of the large size they have attained by the time they are discovered.

McFarland (1936), in his classic analysis of 301 mixed salivary tumours, demonstrated the relationship of recurrence rate to the size of the tumour enucleated, showing that where the tumour was smaller than a lemon, 20 per cent recurred, but where larger than a lemon the recurrence rate was only 10 per cent.

McFarland's views have received authoritative confirmation from two sources in recent years.

Willis (1953) states: "When a tumour is permitted to grow to a large size before its removal, it is then likely to have incorporated the whole of the potentially neoplastic field of tissue, and to have become more completely encapsulated; hence its enucleation is correspondingly less likely to leave behind the seeds of recurrent growth."

Patey and Thackray (1958), following painstaking serial section studies of 15 parotidectomy specimens from cases of primary mixed tumour, demonstrated that such growths were always single and never of multicentric origin, and also that a potent cause of recurrence lay in areas of local infiltration where rapidly proliferating tumour-cells breached the capsule and merged with normal parotid tissue. This phenomenon was, however, only found in small tumours (that illustrated was ¾ in. diameter), and never in large ones, where the tumour was always walled off by fibrous tissue. The authors conclude that this "may account for the often quoted observation of McFarland, that small tumours are more likely to recur than large ones".

Since it is rare for a parapharyngeal mixed parotid tumour to be less than lemon-size, it may be supposed that the recurrence rate following enucleation should not exceed 10 per cent; in the case of 10 such tumours so treated, and followed up for 1–7 years, Morfit (1955) failed to find any recurrence.

There are, therefore, grounds for believing that the local enucleation of mixed parotid tumours with pharyngeal extension might spare the 90 per cent of patients who are not going to suffer from recurrence the not inconsiderable risk of operative damage to the facial nerve which they would otherwise run.

To summarize, it may therefore be said that the advantages of the intra-oral approach to parotid tumours with pharyngeal extension over the external approach with conservative parotidectomy are:—

1. Simplicity of operative procedure.
2. A recurrence rate probably not very much higher than that following total parotidectomy.
3. The elimination of any possibility of damage to the facial nerve (except where a combined interno-external approach has to be used in very large dumb-bell tumours).
4. A later external approach, in the event of recurrence of growth, is not prejudiced, since intra-oral enucleation does not interfere with the parotid tissue in which the facial nerve is embedded.
5. Absence of external scar and of anæsthesia over the distribution of the great auricular nerve.
6. Frey's (auriculo-temporal nerve) syndrome and salivary fistula formation do not occur.

Treatment of Recurrent Growths.—While primary tumours of mixed salivary type appear to arise from a single focus, recurrent growths are commonly multiple (Foote and Frazell, 1953; Ackerman and Wheat, 1955; Patey and Thackray, 1958).

Total conservative parotidectomy using a wide external approach should be undertaken without delay in all cases of recurrent growth following enucleation.

SUMMARY

An account is given of the clinical appearances and diagnosis of 5 cases of parotid tumours with pharyngeal extension. Of the 5 cases, 4 were of mixed salivary type and 1 was a fibroma.

A new oral approach to allow the enucleation of such tumours via the oral cavity is detailed, and its application to the 5 cases recounted.

The advantages of the oral approach when compared with conservative parotidectomy are enumerated.

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Additional thanks are due to Dr. J. L. Edwards, Consultant Pathologist, Royal Hospital, Sheffield, who reported on the histopathology of all growths, and to Mr. A. S. Foster, Medical Artist to the United Sheffield Hospitals, who prepared *Figs.* 322 and 323.

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LARGE GANGLIA OCCURRING IN TENDONS

A DISCUSSION OF THREE CASES

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Little attention has been focused previously on large ganglia found in muscles or tendons. Lecène (1927) described the findings in 1 case of a cyst within the common extensor tendon to the middle finger over the dorsum of the hand. Brooks (1952), in his paper on nerve compression by simple ganglia, illustrated one case of ganglion within the substance of the flexor carpi ulnaris muscle, just below the elbow-joint. Broomhead (1955) discussed the findings in 14 cases of ganglia, 3 of them around the elbow-joint and 11 around the knee-joint. In his series, collected over a period of 24 years, there were only 4 examples resembling the cases about to be described. Graves (1956) reported in this Journal 1 case of a large ganglion in the tendon of peroneus longus below the knee.

Between 1941 and 1958, 3 examples of these ganglia have been seen at the Nuffield Orthopædic Centre, Oxford. One was found in the substance of the extensor carpi ulnaris muscle in the region of the elbow-joint and the other two in muscles about the knee-joint.

These cases appear to be quite uncommon. They are interesting because they may present a problem in clinical diagnosis. Two of our patients were admitted quickly to hospital, on the suspicion that their ganglia were malignant tumours of muscle. Furthermore, a study of the histology in these cases lends support to the theory that ganglia arise, primarily, from degeneration of the ground substance of tendons or connective tissues and not from synovial herniations or remnants.

CASE REPORTS

Case 1.—Mrs. M. T., aged 30 years, was seen on May 21, 1941. Three months prior to admission she had

twisted her right ankle. Subsequently she complained of aching pain below the right knee-joint. This disappeared gradually, but she noted the appearance of a lump below and on the lateral side of the knee-joint. It was slightly tender and appeared to be increasing in size.

She had a rounded lump measuring 1½ in. across, situated in the extensor muscle group at the upper end of the right fibula. It was not attached to skin. It was deep to the deep fascia and it did not move when the regional muscles were contracted. It was tense, but fluctuant.

FIG. 325.—*Case* 1. Ganglion in peroneal muscles.

FIG. 326.—*Case* 2. Ganglion in the extensor carpi radialis longus muscle.

At operation the swelling was exposed through a long incision over the peroneal compartment of the right leg. A pale, longitudinally disposed oval mass was found within the peroneal muscles. Its upper end tapered to a rounded tendon 2 in. long. Below this it formed a 4-in. rounded belly tapering at its lower end to a slender tendon (*Fig.* 325). It was easily separated from the surrounding muscles. Yellow gelatinous material exuded during dissection. There was no demonstrable communication with the knee or superior tibio-fibular joints.

Case 2.—Mrs. H. B., aged 65 years, seen on April 14, 1958. Seven months prior to admission she first noticed a painless swelling in her right forearm, just below the outer side of the elbow-joint. It had continued to increase in size gradually. She complained of an aching sensation in her right wrist, with inability to flex that joint fully.

She had a fusiform non-tender swelling about 4 in. long lying in the extensor muscle group of her right forearm.

At operation this was found to be a large, well-defined, dumbbell-shaped cystic swelling with a tendon at its upper end. The main mass lay in the substance of the extensor carpi radialis longus muscle (*Fig.* 326). There were muscle-fibres stretched over its surface and it was easily separated from the muscle belly. The cyst contained clear gelatinous fluid. No communication with the elbow-joint could be demonstrated.

Case 3.—Mr. J. M., aged 56 years. This man complained of a painful swelling on the outer side of his left leg which had been present for many years. It tended to fluctuate in size. He claimed that pain radiated down the outer side of his leg to the dorsum of his foot.

He was found to have an egg-sized, subfascial, firm swelling in the upper anterolateral compartment of his left leg. There were no objective sensory or motor defects in the leg. The swelling was fluctuant and it could be transilluminated.

At operation a large ovoid swelling, 2 in. × 4 in., was found within the substance of the extensor hallucis longus muscle. There was a stout tendon at its upper end. Muscle-fibres were stretched over it, but the surrounding bellies were easily separated from its surface (*Fig.* 327). The cyst contained clear glairy fluid. There was no communication with the superior tibio-fibular joint or with the knee-joint.

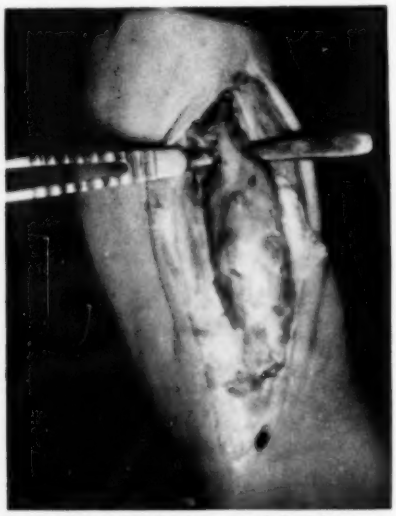

FIG. 327.—*Case* 3. Ganglion in extensor hallucis longus muscle.

HISTOLOGY

Broomhead remarked on the uniform histological picture in 11 of his cases. In fact, there may be considerable variation in the histological appearances of these lesions (as illustrated in King's (1932) paper) and in the terminology applied to their descriptions. It is felt that this indicates a lack of appreciation of the nature of origin and of the evolution of these lesions. In the present series,

Case 1 was reported as a degenerative cyst of tendon, *Case* 2 as a synovial cyst, and *Case* 3 as a bursa in the extensor hallucis longus muscle. However, it is correct to say that these lesions are all identical in that the basic changes of ground substance alteration, cyst formation, collagen fibrillation, and local cellular responses are comparable. These changes evolve to varying stages in each case, thus accounting for the diversity of appearances between different specimens. Some present as unilocular cysts, others as multilocular cysts with a greater preponderance of round-cell infiltration in their walls. Our findings in these three cases are in agreement with the conclusions drawn by Delarue, Miguel, Payen, and Roussel (1955), and Delarue, Miguel, and Civatte (1957) on the pathogenesis of ganglia.

DISCUSSION

The pathogenesis of ganglia in general has been subject to discussion for many years. In 1850 Gosselin presented a careful study on synovial cysts of the hand and wrist. Of their origin he wrote: "La science est loin d'être bien fixée sur ce point." The same has been said in recent years (Brooks, 1952), though now the problem appears to be nearer final solution.

Gosselin referred to four theories current at that time, though he dismissed all of them in favour of his own, which itself did not survive. The first and oldest suggested that these cysts developed specifically from the cells of the tissues; the second, and most popular, that they arose as herniations of synovial membrane from joints or tendon sheaths; the third claimed that cyst formation resulted from the escape of synovial fluid following rupture of synovial sheaths; and the fourth theory dealt with a vague concept of *hydropisie*. Carp and Stout (1928) concluded that ganglia are cysts resulting from mucinous degeneration of connective tissue. Although they had not seen these lesions occurring within tendons, they referred to their existence. De Orsay, Mecray, and Ferguson (1937), using Mallory's phosphotungstic acid stain in studying their sections of ganglia, concluded that a specific degeneration of collagen fibres could be demonstrated.

Lecène (1927) described one case of a ganglion occurring within the common extensor tendon to the middle finger. Other cases have been mentioned above. Recent works can be correlated with their interpretations and so, at last, the problem of pathogenesis can be resolved to a point where it seems that ganglia arise primarily from a defect in the ground substance of connective tissues, joint capsules, or tendons.

Delarue and others (1955) reviewed the developments on the study of tendon structure. They drew attention to the importance of the ground substance as the "milieu-intérieur" for the nourishment of the fibres and cells. Using a variety of histochemical methods they were able to show that ground substance alterations occurred constantly in various lesions, such as ganglia, 'spontaneous' tendon ruptures, and certain tumours of tendons, or aponeuroses. In certain cases, they said, these ground substance alterations gave rise to the formation of cavities full of a viscous or myxoid substance.

Writing again in 1957, Delarue and co-workers claim to have demonstrated abnormalities in the glucosidic fraction of the ground substance, though they say that changes in the various protein components still remain beyond the scope of critical histochemical analysis. These changes lead to collagen fibre dedifferentiation and fragmentation, with varying local cellular responses which evolve slowly, and it is in this way that ganglia are formed.

SUMMARY

1. Three cases of large ganglia occurring in muscles and tendons are described.

2. The pathogenesis of ganglia is discussed in some detail.

3. A plea is made for uniformity in description.

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DERMATOPATHIC LYMPHADENITIS (LIPOMELANIC RETICULOSIS) SIMULATING MAMMARY CARCINOMA

By J. A. DOSSETT and H. S. SHUCKSMITH

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THE association of a generalized skin eruption with enlarged superficial lymphatic glands is well known. The condition appears to have been originally described by Jadassohn in 1892, and since then various terms have been applied to the lesions. 'Lipomelanic reticulosis' (Pautrier and Woringer, 1937) is one of the more generally accepted names for the glandular lesion. This descriptive term refers to the presence of melanin and stainable fat, usually to be found in the areas of histiocytic proliferation in the lymphatic glands. Plasma cell and eosinophilic infiltration are also to be seen. As the presence of lipoid and melanotic pigment is not a constant feature, Hurwitt (1942) suggested 'dermatopathic lymphadenitis' as a more exact term. In this he was supported by Jarrett and Kellett (1951), who found melanin to be absent in 3 of their 16 cases.

The skin lesions are of diverse types. For example, pityriasis rubra, pityriasis rubra pilaris, polymorphic prurigo, flexural seborrhœic dermatitis, allergic eczema, neurodermatitis, and exfoliative dermatitis are but a few of the many diagnoses illustrating the wide variety of skin lesions. More complete lists can be obtained from the papers of Pautrier and Woringer (1939), Agress and Fishman (1950), Obermayer and Fox (1949), and Laipply (1948). It is generally held that dermatopathic lymphadenitis can arise from any chronic pruritic dermatosis (Jarrett and Kellett, 1951) and these authors further hold that the dominant lesion is a generalized erythrodermia, with superimposed degrees of exfoliation, lichenification, pigmentation, excoriation, and low-grade sepsis.

Clinically dermatopathic lymphadenitis must be differentiated from other lymphadenopathies and particularly from such conditions as Hodgkin's disease, which may be associated with a skin lesion, and mycosis fungoides. Many of the accounts of lipomelanic reticulosis progressing to a malignant lesion are not well authenticated. It appears, however, that any of the reticuloses, e.g., monocytic leukæmia or lymphosarcoma, may occur independently in a patient with dermatopathic lymphadenitis, and there is no definite evidence in these cases that lipomelanic reticulosis is itself a pre-malignant disease. Lymph-node biopsy may well be necessary to exclude two coincident conditions. Other causes of enlarged glands which have led to mistaken diagnoses are tuberculous lymphadenitis and sarcoidosis.

Histologically the lesions most likely to be confused are Hodgkin's disease—owing to the histiocytic hyperplasia and eosinophilia—giant follicular lymphoma, and simple sinus catarrh. Differentiation of these lesions is adequately covered in the excellent paper of Jarrett and Kellett.

The present case is reported because of the very unusual presentation which led to the possibility of the mistaken diagnosis of carcinoma of the breast with secondary axillary gland involvement. We have been unable to find a similar case in the literature.

CASE REPORT

The patient was a married woman of 37 years who worked as a kitchen help. She presented with a 3-week history of a lump in the left breast which she had first noticed while washing herself. The lump was neither painful nor tender and there had been no discharge from either nipple. She had two children, now 10 years and 5 years old, and an incomplete abortion had been evacuated 11 years previously. The first child had been suckled for two months when an abscess had developed in the right breast. This had failed to resolve and surgical excision was undertaken at Leeds General Infirmary. Histological examination confirmed the clinical diagnosis. Her second pregnancy was uneventful and the child was suckled for 10 months.

She had suffered from flexural eczema for several years. The disease varied in intensity and was at present in a relatively quiescent phase. It had been troublesome, with intense irritation, about three months previously.

ON EXAMINATION.—A lump about the size of a pea was palpable 3 cm. lateral to the nipple. It was hard, discrete, mobile, and was not attached to skin or deep structures. There were no signs of inflammation and the lump was not tender. Enlarged glands were palpable in both axillæ, but they were larger on the homolateral side.

The skin lesions were best marked on the flexural surfaces of the limbs, but were also seen on the abdomen. There was thickening of the skin and lichenification. Excoriation was minimal and sepsis was not apparent. No other abnormalities were found on physical examination. In the peripheral blood the total white cell count was 8800 per ml. with 6 per cent of eosinophils.

The clinical diagnosis was thought to rest between fibrocystic disease of the breast and carcinoma. As is customary in this clinic with patients of this age, mastectomy was advised in preference to biopsy. Simple mastectomy with removal of one of the enlarged glands was undertaken. During the operation a 'milk-like' fluid exuded from the nipple.

PATHOLOGY.—The breast weighed 300 g., and 3 cm. lateral to the nipple and lying in a flask-shaped depression in the breast tissue was a lymphatic gland 1·5 cm. in diameter. This was the lump felt in the breast before operation. A lymph-node 2 cm. in diameter was attached to the axillary tail. On cutting the breast before fixation a pale 'milk-like' fluid escaped from dilated ducts leading up to the nipple. No localized lesion was found. Blocks for histological examination were taken from several areas of the breast.

Histology.—Both lymphatic glands showed a massive diffuse histiocytic or reticulum-cell hyperplasia, in which the usual structure of the lymphoid follicles was partly obliterated. These reticulum cells possessed regular nuclei and mitotic figures were not seen. Some of the cells contained melanin (*Fig.* 328), and pigmentation was particularly prominent in the region of the peripheral lymph-sinuses. The Prussian blue reaction for iron was negative. Many of the histiocytes had a vacuolated cytoplasm, but with Sharlach R stained frozen sections fat could be demonstrated in only a small number of the cells. Lymphocytes were reduced in numbers and formed a thin rim round the germinal centres. Eosinophils were scattered through the glands. This is the picture of lipomelanic reticulosis.

The breast showed the widespread lesions of mammary duct ectasia (Dossett, 1958) and dilated ducts were found reaching from the nipple to the periphery of the gland.

In hæmatoxylin and eosin stained sections they contained a homogeneous pink secretion. There was no evidence of an inflammatory lesion inside or outside the ducts. Lobules dependent on the dilated ducts were either involuted or showed severe distortion. Some normal ducts were interspersed between the dilated ducts and their associated lobules were normal. There was no epithelial hyperplasia to suggest either premalignant or malignant change.

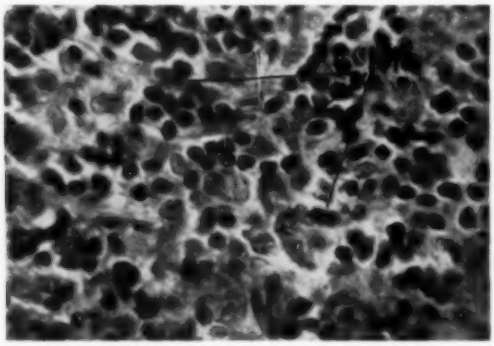

FIG. 328.—Lipomelanic reticulosis, showing melanin containing cells (M) and histiocytic proliferation in a peripheral lymph-sinus.

At follow-up a year later the woman was quite well apart from the persistent skin disease.

DISCUSSION

This case illustrated yet another pitfall in the clinical diagnosis of the lump in the breast. It is well known that lymphatic glands may be found embedded in the female breast. In our experience they are frequently seen at the tip of the axillary tail, but in a study of over 350 breasts undertaken for another purpose, a lymphatic gland was found in the remainder of the breast on only one other occasion. The rarity with which lymphoid tumours are confused with breast tumours must in part be due to this lack of lymphatic tissue.

It is unusual for glandular enlargement to persist in dermatopathic lymphadenitis after the skin lesion has entered a quiescent phase. Although the glands usually become smaller and histologically revert to a normal pattern, this is not always so, and Jarrett and Kellett (1951) quote a case which, during a remission and while free from irritation, had grossly enlarged axillary glands. In our case this undoubtedly added to the difficulty of diagnosis.

Mammary duct ectasia may be associated with enlarged axillary nodes, particularly when periduct inflammation—the so-called plasma cell mastitis of Ewing—is present. In a study of 75 cases of this disease (Dossett, 1958) 26 showed associated periduct inflammation of varying degree. Although the axillary glands were examined on 49 occasions, no lesion resembling lipomelanic reticulosis was seen. Many of these glands showed a simple reactive hyperplasia, but melanin was not observed, histiocytic hyperplasia was not so extreme, and eosinophils were rarely found. Lymph-follicles were, however, more prominent. There is little likelihood of confusing the histological appearances in the two conditions, but clinical differentiation when a skin lesion and duct ectasia coexist may be impossible.

SUMMARY

A case of dermatopathic lymphadenitis presenting as a lump in the breast and enlarged lymphatic glands is described. The mass in the breast was an enlarged lymphatic gland.

The skin lesion was in a quiescent phase and diagnosis was made on histological examination of an axillary lymph-node and the lymph-node embedded in the breast.

Reference is made to a survey of 350 breasts in which lymph-nodes were found in the main part of the breast on only 2 occasions.

The differentiation of enlarged lymphatic glands due to duct ectasia from those due to lipomelanic reticulosis is discussed.

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SHORT NOTES OF RARE OR OBSCURE CASES

LONG SURVIVAL AFTER EXTENSIVE RESECTION OF URINARY TRACT FOR TRANSITIONAL-CELL CARCINOMA

REPORT OF A CASE IN A WOMAN AGED 44 YEARS

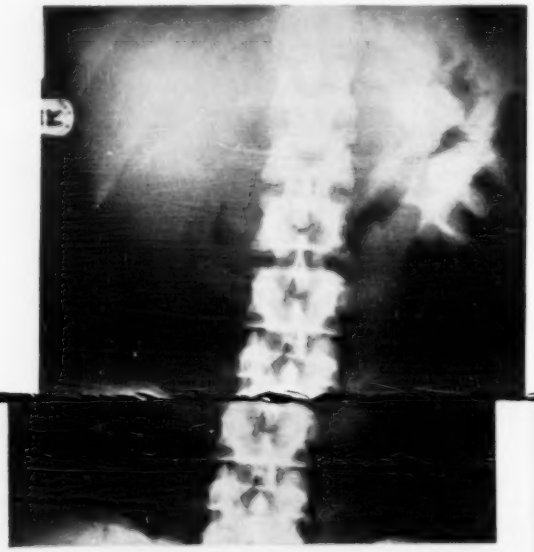
By GWYN MORRIS, Cardiff

Transitional cell carcinoma is commonplace. The following account is worthy of record because of the following features:—

1. Because of the apparently hopeless prognosis when first seen owing to the extent of the lesion.
2. Many years of normal activity after radical surgery.
3. Use of deep X-ray therapy for papillomatosis of the renal pelvis in a surviving kidney.

CASE REPORT

A woman aged 44 years referred by a colleague (Mr. H. O. Jones) was first seen in August, 1952. She gave a history of difficulty in micturition for the preceding twelve months associated with dysuria and hæmaturia, together with lower abdominal pain relieved on micturition. These latter symptoms had been present for over six months. Difficulty in micturition became more pronounced, and for the four weeks prior to her admission to hospital she had defæcation each time with strain on micturition. She also complained of severe anorexia and had lost 2 st. in weight.



A

B

Fig. 3.—A, Enlarged bladder with vague outline of filling defect and left-sided hydro-ureter and hydronephrosis. B, Drawing of appearances at original presentation.

On Examination.—There was a chronic retention with overflow. The bladder was enlarged half-way to the umbilicus, and vaginal examination revealed extensive anterior infiltration with no obvious fixation of the vaginal walls. A nodular growth projected into the right fornix. The uterus was retroverted and normal.

Catheter specimen of urine contained some albumin with numerous leucocytes and moderate numbers of red blood-corpuscles. Urine culture revealed a moderate growth of enterococcus. The blood-count showed a marked hyperchromic anæmia, erythrocytes 3,090,000, white blood-cells 10,300. The hæmoglobin was 57 per cent of normal and the blood-urea 47 mg. per cent.

The intravenous pyelogram showed an enlarged right renal outline with no concentration of the dye in the right kidney. There was a severe degree of left hydronephrosis and left hydro-ureter. The bladder was seen to be enlarged with a very vague outline of filling defect (*Fig.* 329 A).

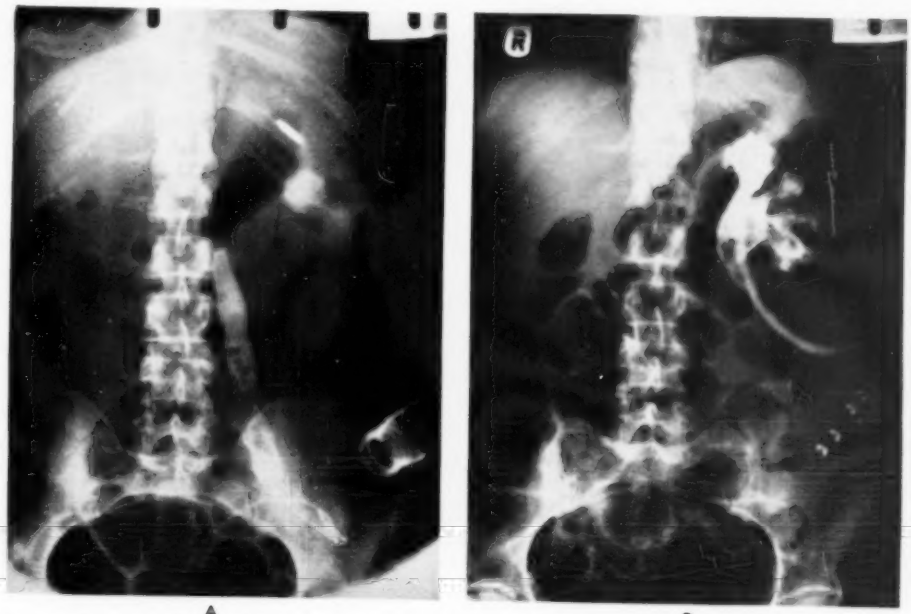
Cystoscopic examination showed the distal half of the bladder to be filled with necrotic tumour involving the right half of the trigone, the right ureteric orifice, and the whole of the internal meatus (*Fig.* 329 B). Radiography of the lung fields showed no pulmonary secondary deposits.

AT OPERATION.—Following blood transfusion and through a transverse suprapubic incision a total cystectomy with bilateral cutaneous ureterostomy was carried out, when it was found that the carcinoma had extended into the lower 5 cm. of the right ureter. Owing to the degree of hydro-ureter on the left side, ureterocolic anastomosis was considered to be inadvisable. The adherent anterior vaginal wall was also resected at the same time, the terminal end of the left ureter being brought out along a small skin flap fashioned to form a nipple. Thick pus drained immediately from the right ureter.

Section report on the bladder confirmed that the tumour was a transitional-cell carcinoma with very little infiltration of the bladder musculature. The tumour did not show much mitotic activity.

Thick pus continued to pour from the right ureter. The right kidney was enlarged and tender. The patient continued to be hyperpyrexial and toxic. Accordingly, 17 days after the cystectomy, a pyonephrotic right kidney was removed together with the remainder of the right ureter.

PROGRESS.—Following this operation she had an uncomplicated convalescence and she was discharged 2 weeks



A B

FIG. 330.—A, Left retrograde pyelogram carried out via the cutaneous ureterostomy. B, Extensive generalized negative filling defects in retrograde pyelogram.

FIG. 331.—Retrograde pyelogram performed in September, 1958.

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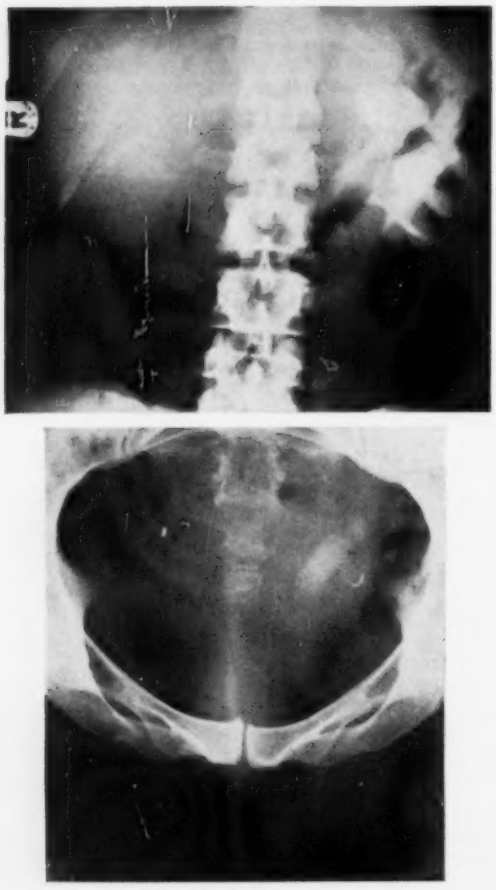

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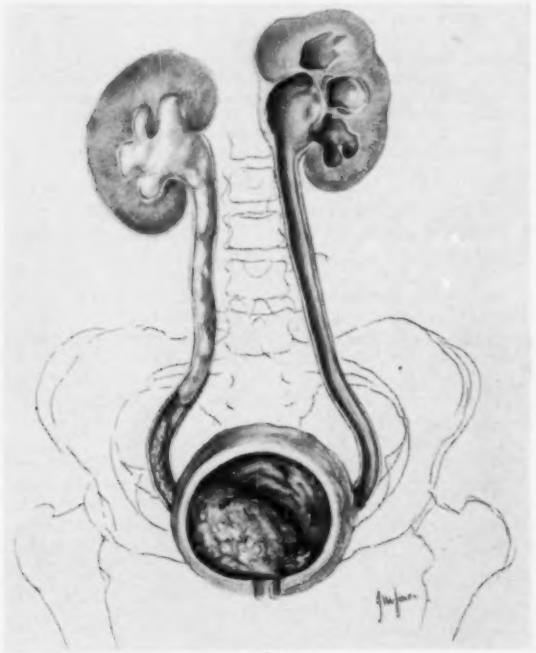

B

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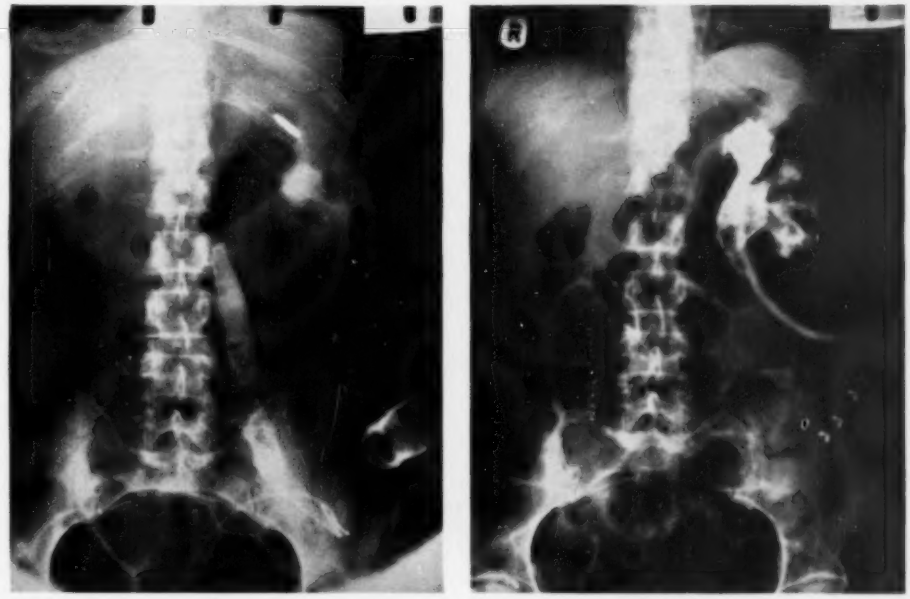

A B

FIG. 330.—A, Left retrograde pyelogram carried out via the cutaneous ureterostomy. B, Extensive generalized negative filling defects in retrograde pyelogram.

FIG. 331.—Retrograde pyelogram performed in September, 1958.

after the nephrectomy. The cutaneous ureterostomy was working well, the blood-urea was normal, and she was already putting on weight.

This patient remained fit and well with no clinical or radiological evidence of metastases. She continued to put on weight and there was no recurrence of anæmia. In February, 1956, 3½ years after operation, she was re-admitted to the medical unit (Professor Harold Scarborough) with a history of lassitude and extreme anorexia of 5 weeks' duration associated with severe headaches, nausea, and vomiting. In addition, she complained of oliguria and œdema of the ankles for the two weeks prior to admission to hospital. Her tongue was brown, furred, and dry and she was obviously uræmic. The blood-urea was 428 mg. per cent and the serum potassium 9·4 mEq./litre. Following intensive treatment with resonium 'A' 30 g. daily, a left retrograde pyelogram was carried out via the cutaneous ureterostomy. The appearances are as shown in *Fig.* 330. There was a marked filling defect involving the junction of the lower and mid-thirds of the left ureter. A little of the dye which got into the left kidney showed a marked hydrocalycosis.

SECOND OPERATION.—Accordingly, the left ureter was exposed well above the growth and a cutaneous ureterostomy performed at a higher level. Section of the ablated ureter showed a transitional-cell carcinoma of the ureter with invasion of a blood-vessel at one point.

There was an immediate and satisfactory improvement in urinary output with a marked fall in the blood-urea. The serum potassium fell to normal within one week. The hæmoglobin, however, remained at 34 mg. per cent. Retrograde pyelography was then carried out through the new ureterostomy opening. This showed extensive generalized negative filling defects in the pelvis and major calices (*Fig.* 330 B). Some papilliferous debris together with blood-clot continued to come down the stump of the ureter which remained, and in April, 1956, deep X-ray therapy (Dr. Glyn C. D. Evans) was carried out to the whole of the left kidney.

The patient was discharged home wearing a ureterostomy plastic cup.

For the past 2½ years this patient has remained very fit and well. The blood-urea is still abnormal (about 100 mg. per cent), but there is no anæmia. She has now resumed teaching duties in school. The only evidence of any new development in the papillomatosis was when a subcutaneous nodule the size of a hazel nut was excised under a local anæsthetic. This was present at the extreme terminal end of the original left ureterostomy at the mucocutaneous junction, and in all probability must have developed in a minute portion of ureteric mucosa left after the original ureterectomy at this site. Section report on this tumour showed a transitional-cell papilloma with no invasion of the base.

Fig. 331 shows a retrograde pyelogram carried out in September, 1958.

METASTATIC CARCINOMA OF THE SPERMATIC CORD

By J. L. WILLIAMS

SENIOR SURGICAL REGISTRAR, ROYAL HOSPITAL, SHEFFIELD

SECONDARY involvement of the spermatic cord from a primary lesion in the gastro-intestinal tract is very rare.

Previous cases have been described by Lewis, Goodman, and Randall (1944) and Jones Williams and Thomas (1955), both from a carcinoma of the stomach. Willis (1952) thought that the third case described by Debernardi (1907) was probably a secondary growth from a carcinoma of the colon and not primary as described by the author. A further case is presented here.

CASE REPORT

H. B., aged 62 years, a press operator, was admitted in April, 1958, complaining of a painful lump in the right scrotum which had been present for several weeks and was increasing in size. He had previously been investigated in 1955 for dysuria, but intravenous pyelography and cystoscopy showed no abnormality. It was noted at that time that he had a small right hydrocele.

He still had some frequency of micturition but no hæmaturia. His bowel action was regular and he had a good appetite. He had a cough which had been present for some years and brought up a little yellow sputum each morning, but no blood.

ON EXAMINATION.—He was a fairly fit man. He had a left paramedian abdominal scar. The liver and spleen were not palpable, there were no masses, and rectal examination showed only slight enlargement of the prostate. The left testis, epididymis, and cord were normal. On the right side he had a small hydrocele, but the underlying testis felt normal. The distal third of the spermatic cord was extremely hard and thickened to a diameter of about 1 cm. This thickening extended to the upper pole of the epididymis, the remainder of which felt normal. The cord was tender and the testis was lying higher in the scrotum than on the left side. The chest radiograph was normal and urine examination showed no abnormality. No enlarged lymph-nodes were found.

AT OPERATION (April 10, 1958).—Through an incision in the right groin the inguinal canal was opened. The proximal part of the cord felt quite normal and the physical signs noted above were unchanged. A right orchidectomy was performed with an uneventful recovery.

PATHOLOGY (Dr. J. L. Edwards) (*Figs.* 332, 333).—"From the right testis.

"*Macroscopic.*—Testis with epididymis 5 × 4 × 4 cm. and 8 cm. attached cord. Cross section: In the region of the epididymis is a cavity 3 × 1·8 × 1 cm.; its wall is white, hard, and firm and is 1 cm. at its greatest thickness. The contents of the cavity are green and mucoid. The cord is to one side of the firm white tissue which extends along the cord for a distance of 4 cm.

"*Microscopic.*—The firm white tissue, in my opinion, is infiltrated by mucinoid adenocarcinoma—poorly differentiated. The neoplasm is prominent in perivascular and perineural lymphatics. I see no evidence of primary neoplasm in the testis. It seems probable that the neoplasm has travelled by the lymphatics from either stomach or colon. The neoplasm does not appear to be present in the proximal end of the vas given to us."

PREVIOUS HISTORY.—In December, 1957, the patient had been admitted to hospital with a subacute intestinal obstruction, the symptoms of which had been present for the previous five weeks. At laparotomy on Jan. 2, 1958 (Mr. D. H. Randall), a stenosing growth in the terminal ileum, 6 in. from the ileocæcal valve, involving the serous coat, was found. There was a hard nodule on the cæcal wall in which the tip of the appendix was embedded. There were two nodules on the mesentery of the small bowel, but no palpable lymph-nodes. The liver was normal and no other abnormality was found after a careful

search, as these lesions were thought to be metastases from a primary growth elsewhere in the abdomen. A right hemicolectomy was performed with an uneventful recovery. Apart from the right hydrocele no abnormality of the spermatic cord was found on this admission.

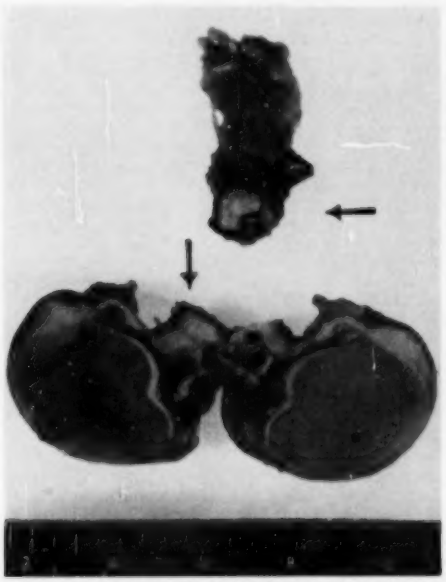

FIG. 332.—The spermatic cord, testis, and epididymis cut across to show involvement by growth.

PATHOLOGY (Dr. R. A. Caldwell).—Right hemicolectomy specimen.

"There was a hard mass 2 cm. in diameter, 12 cm. from the ileocæcal valve on the mesenteric border of the ileum. This mass partially surrounded and constricted the lumen but was not in continuity with the mucosa.

"There was another hard mass 1·5 cm. in diameter firmly fixed to the cæcal wall but not involving the mucosal coat. This mass had a yellowish centre and was continuous with the tip of the appendix. There were no obvious lymph-nodes in the mesentery."

Histology.—"The two masses described above have the structure of an anaplastic mucin-secreting adenocarcinoma with infiltration of the mesentery of the small bowel.

"Two other nodules which were obtained separately also showed infiltration by a mucin-secreting adenocarcinoma." (They had been attached to the mesentery of the small bowel.)

The patient was quite well until June, 1958, when he was admitted to another hospital with severe headaches and vomiting. He died in July, 1958, and unfortunately no autopsy was obtained.

DISCUSSION

The exact site of the primary growth is difficult to determine. The pathological appearances indicate a gastro-intestinal growth. No lesion was found in the stomach at laparotomy, but this does not entirely rule out the possibility. The appendix could have been the site of the primary growth. In the previously reported cases of metastatic carcinoma of the spermatic cord, the spread of tumour cells was thought to be either lymphatic (Jones Williams and Thomas, 1955), directly from peritoneal deposits (Lewis, Goodman, and Randall, 1944), or retrogradely by involvement of the spermatic vein (Debernardi, 1907). This latter method has been observed several times from a primary lesion in the left kidney (Willis, 1952).

In this case there was no evidence of involvement of the spermatic veins, and a normal cord between the peritoneum and metastasis rules out a direct spread.

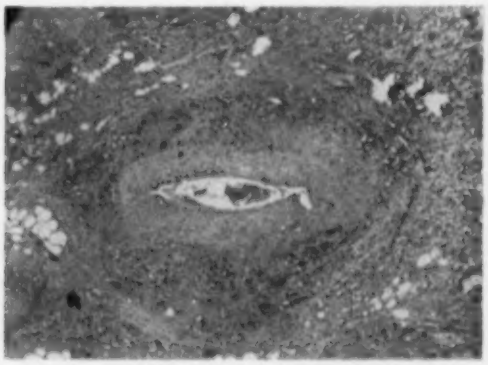

FIG. 333.—Showing perivascular lymphatic metastases in the spermatic cord. (× 18.)

The lymphatics in the cord could have been involved from the secondary growth, but retrograde lymphatic spread seems the most likely method of involvement of the cord. Although at laparotomy no obvious involvement around the spermatic vessels was noted, the close proximity of these to the growth attached to the cæcum and appendix suggests the probable method of involvement of the lymphatics.

SUMMARY

A secondary mucin-secreting adenocarcinoma involving the spermatic cord is described, which was thought to be due to retrograde lymphatic spread.

The exact site of the primary growth was not found, but could have been from the appendix.

I wish to thank Mr. J. C. Anderson for permission to publish this case; Mr. D. H. Randall for permission to use his case notes; Dr. J. L. Edwards for the pathology report on the spermatic cord; Dr. R. A. Caldwell and the Department of Pathology, University of Sheffield, for the pathology report on the intestinal tumours, and the Department of Medical Photography, Sheffield United Hospitals, for the photographs.

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CHORIONEPITHELIOMA TERATOMA OF TESTIS

BREAST HYPERTROPHY PRECEDING PALPABLE TESTICULAR TUMOUR

By P. W. MONCKTON COPEMAN, London

This case history is presented because breast hypertrophy preceded by more than three months the palpable scrotal swelling in a patient with a teratoma of the testis of the chorionepithelioma type. This case is thought to be unique in that the gynæcomastia (itself a rare accompaniment of testicular neoplasms) was not associated with metastases.

CASE REPORT

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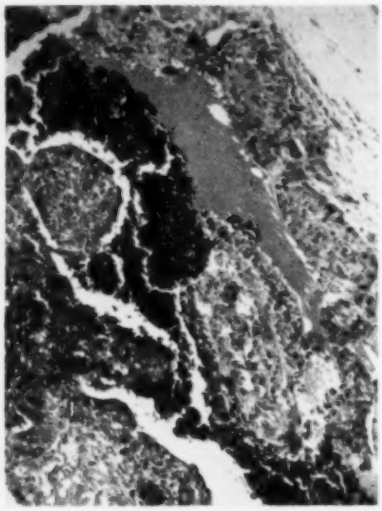

Fig. 334.—Chorionic villus in blood sinus. (× 140.)

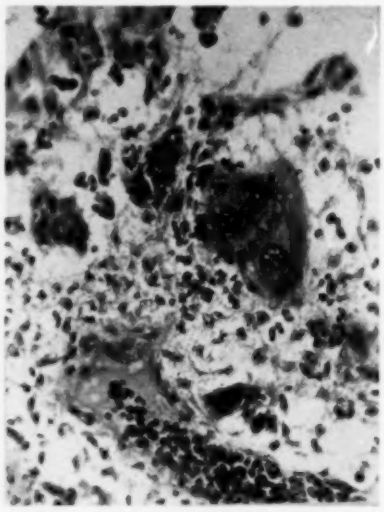

Fig. 335.—Island of syncytium. (× 280.)

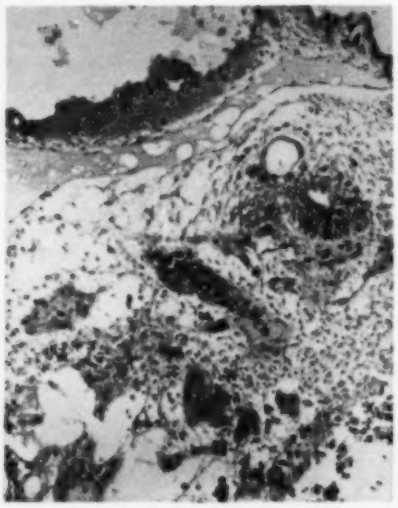

Fig. 336.—Syncytial mass. (× 140.)

Fig. 337.—Adenocarcinomatous-tubule formation. (× 140.)

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A tense translucent hydrocele the size of a tangerine was found in the right scrotum through which the testis could not be palpated. Some degree of testicular sensation was still present. The left testis and both spermatic cords were normal. The breasts were the size of half a squash ball—noticeably enlarged for a patient who was not fat. No discharge could be expressed, but in the mornings a small amount of what appeared to be dried secretion was noted on both nipples. Body hair was normally distributed. No masses were felt in the abdomen, the liver was impalpable, and there was no lymphadenopathy. No secondary deposits were seen on the chest radiograph. The Hogben test was negative.

An orchidectomy was performed on Nov. 6, 1954 (Mr. R. H. O. B. Robinson). Post-operatively the patient received deep X-ray therapy to the region of the para-aortic and iliac lymph-nodes, a total dosage of 3000 r. being given over 4 weeks in 20 divided doses. The testis on section was twice the normal size and largely replaced by tumour tissue, parts of which had the characteristic histology of a chorionepithelioma (Dr. R. H. Thomlinson) (*Figs.* 334–337). The patient is alive and fit four years after the operation, and there is still no clinical or radiological evidence of metastasis.

Comment.—A teratoma which has differentiated into chorionepithelioma type of tissue is diagnosed by identifying the cell arrangement of the syncytium (*see Figs.* 335, 336) and the Langhans and chorionic wandering cells (Handfield-Jones, 1926). In this case the chorionic villi were readily distinguishable (*see Fig.* 334). The incidence of chorionepithelioma of the testis in the larger published series of testicular tumours varies from 2·5 per cent to 6·4 per cent (Friedman and Moore, 1946; Lewis, 1948; Gray, McDonald, and Thompson, 1950; Schwartz and Mallis, 1954; Thomas and Bischoff, 1954). The 'pure' primary chorionepithelioma of the testis unassociated with other histological elements is excessively rare. The tumour under discussion contained a number of other readily identifiable formations, among them the adenocarcinomatous tubules seen in *Fig.* 337.

Gynæcomastia associated with testicular tumours is not common—approximately 7 per cent in most series (*see above*). Some of these were teratomata of the chorionepithelioma type, though breast enlargement is not confined to this variety of tumour. Often the gynæcomastia coincides with the appearance of secondary deposits. In the present patient no metastases were found and none has appeared four years after orchidectomy and deep X-ray therapy. The gynæcomastia regressed rapidly following operation and has not returned. Rarely does the patient survive more than two years, except in those cases in which no metastases are demonstrated before operation and in which orchidectomy is combined with post-operative radiation to the abdominal lymph-nodes (Whittle, 1957).

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CHORIONEPITHELIOMA TERATOMA OF TESTIS

BREAST HYPERTROPHY PRECEDING PALPABLE TESTICULAR TUMOUR

By P. W. MONCKTON COPEMAN, London

THIS case history is presented because breast hypertrophy preceded by more than three months the palpable scrotal swelling in a patient with a teratoma of the testis of the chorionepithelioma type. This case is thought to be unique in that the gynæcomastia (itself a rare accompaniment of testicular neoplasms) was not associated with metastases.

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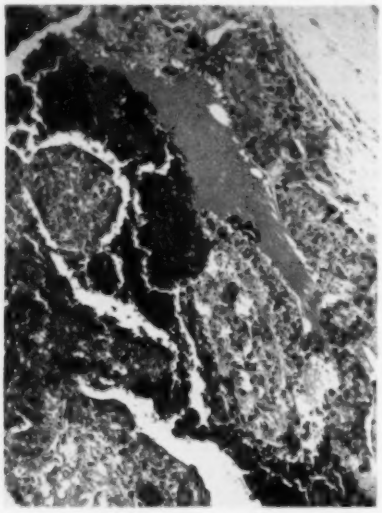

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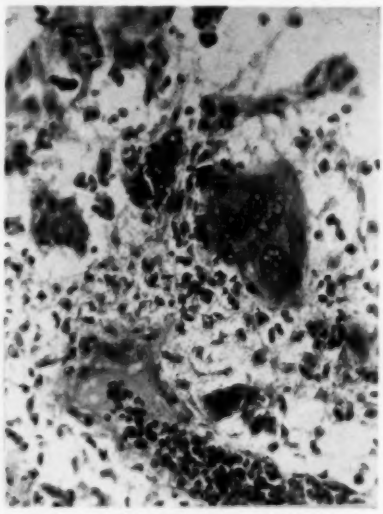

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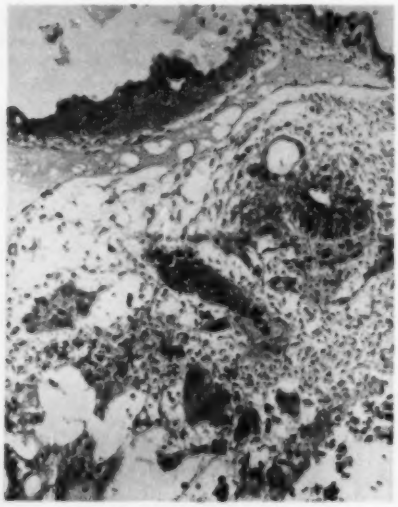

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INTERNATIONAL UNION AGAINST CANCER

CLINICAL STAGE CLASSIFICATION FOR MALIGNANT TUMOURS OF THE BREAST

INTRODUCTION

1. The International Union against Cancer has, since 1950, devoted special attention to the problem of clinical stage classification of malignant tumours.

2. Initially, consideration of this problem was undertaken by the Committee on Tumour Nomenclature and Statistics, under the chairmanship of Dr. Isabella H. Perry (1950–54). This Committee adopted as a basis for its work on clinical stage classification the general definitions of local extension of malignant tumours suggested by the W.H.O. Sub-Committee on the Registration of Cases of Cancer as well as their Statistical Presentation.*

3. In July, 1953, in Copenhagen, the Committee on Tumour Nomenclature and Statistics held a joint meeting with the Committee on Stage-Grouping in Cancer and Presentation of the Results of Treatment of Cancer of the International Congress of Radiology, at which an agreement was reached on a general technique for staging and on a provisional draft for the clinical staging of cancer of the breast, cervix uteri, and larynx. The Joint Committee invited the International Union against Cancer to continue this work with the object of:—

a. Drafting a precise classification for cancer of the breast, cervix uteri, and larynx;

b. Extending the general technique to cancer of certain other sites.

4. The International Union against Cancer accepted this responsibility and, in 1954, the Research Commission of the U.I.C.C. set up a special Committee on Clinical Stage Classification and Applied Statistics, under the chairmanship of Dr. P. F. Denoix, to pursue continuing studies in this field.

5. During the years 1954–58, the Committee prepared two series of draft proposals relating to the general principles to be considered in the clinical stage classification of malignant neoplasms and the application of these principles in particular to breast and larynx. These draft proposals were widely circulated internationally, through the members of the Committee, to secure a broad expression of specialist opinion and comment. On the basis of the suggestions received, revised draft proposals were prepared and again circulated for comment.

6. A special meeting of the Committee was held in Paris in March, 1958, to discuss the revised draft and the suggestions which had been offered. At this meeting, many alternative suggestions and amendments to the Committee's draft report were considered at length and a formal report was prepared for submission to the International Union against Cancer at a meeting held during the VIIth International Cancer Congress in London in July, 1958.

7. The U.I.C.C. took the following decisions regarding malignant tumours of the breast:—

a. All members of the Congress and other interested parties should be invited to send comments to the Chairman of the Committee, not later than March 31, 1959.

b. The Committee should meet later in 1959 and, following consideration of the comments, should, if necessary, revise the proposals.

c. This revised classification should be published in the appropriate medical journals on behalf of the International Union against Cancer.

d. The classification should be brought into clinical use on January 1, 1960, for a trial period of five years.

e. Following this period of appraisal the classification might be further amended, if this is considered necessary, and the final proposals submitted to W.H.O.

8. A second meeting of the Committee was held in Paris in July, 1959, to consider the further comments received and, in particular, to prepare revised proposals for the clinical stage classification of malignant tumours of the breast for publication and trial.

9. Between 1950 and 1959, the members of the Committee have been:—

BACLESSE, F.—Paris
BARAJAS-VALLEJO—Mexico D.F.
BUCALOSSI, P.—Milano
COPELAND, M. M.—Washington D.C.
DENOIX, P. F.—Paris (*Chairman*), 1954
FISCHER, A. W.—Kiel
HAMPERL, H.—Bonn
HARMER, M.—London
HULTBERG, S.—Stockholm
LIMA-BASTO, E.—Lisbon
LOGAN, W. P. D.—London
McWHIRTER, R.—Edinburgh
PERRY, I.—San Francisco (*Chairman*, 1950–54)
RACOV, A. I.—Leningrad
ROXO-NOBRE, M. O.—São Paulo (*Secretary*)
SELLERS, A. H.—Toronto (*Secretary*)

DESCRIPTION OF THE EXTENT OF DISEASE

THE T.N.M. SYSTEM

1. 'Extent of disease' is a complex which can be considered in terms of three components:—

First, the primary tumour designated by the letter T.

Second, the regional lymph-nodes designated by the letter N.

Third, distant metastasis designated by the letter M.

2. Description of the extent of disease within each of these three components is itself complex. Description of the primary tumour (T.), for example, involves consideration of several elements in local extension, the number of which will vary with each site (e.g., in breast—skin, muscle, chest wall). For this purpose, such variations or degrees of local

* W.H.O. Technical Report Series, No. 53, July, 1952.

extension, fully described, can be designated by numbers (e.g., T.1, T.2, etc.). The same procedure may be used for the clinical condition of the regional lymph-nodes (N.) and distant metastases (M.).

3. This system provides for the precise description, identification, and coding of all the various components in the clinical-extent of the disease. From these elements (T., N., M.) clinical stage-grouping can be achieved.

General Rules Common to all Sites of Cancer.—

4. The procedure outlined is intended primarily to apply to cases not previously treated.

5. All cases examined are, without exception, to be identified by T., N., and M. categories. These T., N., M. categories are to be determined at examination prior to any treatment and are to remain unchanged. The examination method used will be general physical examination, roentgen examination, and examinations by instruments available for clinical diagnosis. Evidence obtained at operation is not included. It is important that supplementary provision be made to indicate the findings on histological examination and to denote those cases in which the clinical diagnosis and involvement of nodes is or is not confirmed thereby.

6. The proposed technique of description and classification applicable to all sites of cancer involves three steps:—

First, identification of extent of disease by the use of three symbols:—

T.—Extent of primary tumour,

N.—Condition of regional lymph-nodes, and

M.—Distant metastasis.

Second, assignment to each of these three components of a series of numbers to indicate degrees of extension or involvement, e.g., T.1, T.2, etc.

Third, grouping the T.N.M. assignments into a small number of clinical stages.

Supplementary Histological Classification.—

7. In regard to histological information, the following points should be noted:—

a. Histological findings shall not modify the clinical staging.

b. Results of histological examination can be indicated by attaching the symbols '+' (histological examination positive) and '−' (histological examination negative) to the various T.N.M. categories, e.g., N.0+, N.1 −, etc.

c. The precise histological type of the tumour should be recorded with a view to possible tabulation, preferably according to the *Histological Nomenclature of Human Tumours*, U.I.C.C. Acta, 1958, **14**, No. 3.

APPLICATION OF THE GENERAL RULES TO MALIGNANT TUMOURS OF THE BREAST

T.N.M. Categories of Malignant Tumours of the Breast.—

1. The following definitions of the T.N.M. categories for breast are proposed:—

T = *Primary Tumour*

T.1.—Tumour of 2 cm. or less in its greatest dimension.
Skin not involved, except in the case of Paget's disease confined to the nipple.
No retraction of nipple.
No pectoral muscle fixation.
No chest wall fixation.

T.2.—Tumour more than 2 cm. but not more than 5 cm. in its greatest dimension.
or Incomplete skin fixation (tethered or dimpled).
or Nipple retraction (in sub-areolar tumours).
or Paget's disease extending beyond the nipple.
No pectoral muscle fixation.
No chest wall fixation.

T.3.—Tumour more than 5 cm. but not more than 10 cm. in its greatest dimension.
or Skin fixation complete (infiltrated or ulcerated).
or *Peau d'orange* present in the tumour area.
or Pectoral muscle fixation (incomplete or complete).
No chest wall fixation.

Note.—Incomplete pectoral muscle fixation indicates that contraction of the pectoral muscle limits the mobility of the tumour.

Complete pectoral muscle fixation indicates that contraction of the pectoral muscle abolishes the mobility of the tumour.

T.4.—Tumour of more than 10 cm. in its greatest dimension.
or Skin involvement or *peau d'orange* wide of the tumour but not beyond the breast area.
or Chest wall fixation.

Note.—The chest wall includes the ribs, intercostal muscles, and serratus anterior muscle; it does not include the pectoral muscle.

N = *Regional Lymph-nodes*

N.0.—No palpable homolateral axillary lymph-nodes.

N.1.—Homolateral axillary lymph-nodes palpable but movable.

N.2.—Homolateral axillary lymph-nodes fixed to one another or to other structures.

N.3.—Homolateral supraclavicular or infraclavicular lymph-nodes movable or fixed.
or Œdema of the arm.

Note.—Œdema of the arm may be caused by lymphatic obstruction; in such circumstances lymph-nodes may not be palpable.

M = *Distant Metastases*

M.0.—No evidence of distant metastases.

M.—Distant metastases, including skin involvement wide of the breast, involvement of the contralateral lymph-nodes or breast, and all cases with clinical or radiographic evidence of metastases to lungs, pleural cavity, skeleton, liver, etc.

Clinical Stages.—

2. The following clinical staging is proposed:—

Stage I.—Tumour of 5 cm. or less (T.1 or T.2).
Skin fixation absent (T.1) or incomplete (T.2).
Nipple may be retracted (T.2) or Paget's disease may be present (T.1 or T.2).

Pectoral muscle fixation absent (T.1 or T.2).
Chest wall fixation absent.
No homolateral axillary nodes palpable (N.0).
No distant metastases (M.0).

Stage II.—Tumour of 5 cm. or less (T.1 or T.2).
Skin fixation absent (T.1) or incomplete (T.2).
Nipple may be retracted (T.2) or Paget's disease may be present (T.1 or T.2).
Pectoral muscle fixation absent (T.1 or T.2).
Chest wall fixation absent.
Homolateral axillary nodes palpable but movable (N.1).
No distant metastases (M.0).

Stage III.—Tumour of more than 5 cm. in diameter (T.3 or T.4).
or Skin fixation complete (T.3) or skin involvement wide of tumour (T.4).
or *Peau d'orange* present in tumour area (T.3) or wide of tumour (T.4).
or Pectoral muscle fixation incomplete or complete (T.3).
or Chest wall fixation present (T.4).
or Homolateral axillary nodes fixed (N.2).
or Œdema of the arm (N.3).
or Homolateral supraclavicular or infraclavicular nodes movable or fixed (N.3).
No distant metastases (M.0).

Stage IV.—Distant metastases present (M.) regardless of the condition of the primary tumour and regional lymph-nodes.

Stage Grouping.—

3. These four stage groupings are therefore designated by the T.N.M. symbols as follows:—

Stage I.—T.1, N.0, M.0
T.2, N.0, M.0

Stage II.—T.1, N.1, M.0
T.2, N.1, M.0

Stage III.—T.1, N.2 or N.3, M.0
T.2, N.2 or N.3, M.0
T.3, N.0, N.1, N.2 or N.3, M.0
T.4, N.0, N.1, N.2 or N.3, M.0

Stage IV.—Any combination of T. and N. symbols including M.

CLINICAL TRIAL OF THE PROPOSED CLINICAL STAGE CLASSIFICATION FOR MALIGNANT TUMOURS OF THE BREAST

1. The U.I.C.C. recommends that its proposed clinical stage classification for malignant tumours of the breast be subjected to a clinical trial beginning January 1, 1960, for a period of five years. Following this period of appraisal the classification will be further reviewed, amended as necessary, and submitted to W.H.O.

Objectives.—

2. The objectives of the staging trial should be twofold:—

a. To assess the practicability of the proposed system in assigning individual cases to the various T.N.M. categories.

b. To determine how far the proposed system provides a clinical stage classification suitable, both for surgeon and radiotherapist, for the purposes mentioned.

Methodology.—

3. The methodology for the conduct of the clinical trial may, and undoubtedly should, vary from one centre to another. In some instances, if the case-histories contain sufficient complete information on extent of disease, a good deal may be able to be learned by a retrospective study. In other, and probably most, instances, only a prospective study will be possible.

4. A special form may be helpful for recording the data necessary for the T.N.M. assignments.

5. In the conduct of the trial, it is essential that full T.N.M. coding be undertaken and that the prescribed stage-grouping be used, as defined. It is desirable, nevertheless, that special studies involving the regrouping or the subdivision of certain categories should be encouraged.

Special Studies.—

6. Among the particular points, to which it is suggested special attention should be given during the trial period, are the following:—

a. The subdivision of 'pectoral muscle fixation' (in T.3) into two: T.3 (a) incomplete, and T.3 (b) complete.

b. The subdivision of 'homolateral axillary lymph-nodes palpable but movable' (N.1) into two: N.1 (a) nodes not considered to contain growth, and N.1 (b) nodes considered to contain growth.

c. The subdivision of 'homolateral axillary lymph-nodes palpable but movable' (N.1) into two: N.1 (x) node single and N.1 (y) nodes multiple.

d. The subdivision of 'homolateral axillary lymph-nodes fixed' (N.2) into two: N.2 (a) fixed to one another, and N.2 (b) fixed to adjacent structures.

7. Comments are particularly invited on:—

a. The use of four degrees of local extent (T.1, T.2, T.3, T.4) and whether three would suffice.

b. The acceptability of the size groupings used for the primary tumour.

Reports.—

8. Reports of experience in the clinical trials of the proposed clinical stage classification are invited. These may be submitted to:—

Professeur agr. P. Denoix,
Institut Gustave Roussy,
Villejuif – Seine – France.

Copies of the full classification may also be obtained from Prof. Denoix.

REVIEWS AND NOTICES OF BOOKS

Dringliche Chirurgie Beim Säugling und Kind. By Doz. Dr. Dr. H. E. Grewe, Dusseldorf. 9 × 6 in. Pp. 180, with 60 illustrations. 1959. Stuttgart: Georg Thieme Verlag. DM25.

This handbook deals with conditions requiring emergency surgery in neonates and infants. The approach is factual and entirely practical. The account is presented topographically, to a large extent obviating a full index, and goes into details of operative technique where this comes within the scope of general surgery. A terminal section is devoted to anæsthesia, shock, fluid and electrolyte replacement, and drug as well as antibiotic dosages.

This clear and concise little book scores particularly where its dogmatism is based on scientifically established knowledge: this makes much of the terminal section especially valuable. Dogma based on personal experience, however, is bound to produce criticism, even though the format of this book precludes detailed appraisal of methodology. The use of prostigmine in the treatment of ileus seems outdated. The paragraph describing operative treatment of intussusception by resection might have received a different emphasis; it is to be hoped that this sort of procedure is not normally left to the surgical novice.

The price of the book seems rather excessive.

Maladies et Syndromes Rares ou Peu Connus. By A. Aimes. 8¾ × 6¼ in. Pp. 306, with 7 illustrations. 1959. Paris: Masson et Cie. 3000 fr.

The author of this little book has previously published a *First Series of Uncommon or Little-known Diseases and Syndromes* (2nd ed., 1950, Masson). The present volume deals with conditions which were omitted from the First Series or concerning which there is more recent information.

It is in two parts: the first gives brief accounts of more than four hundred diseases or syndromes, arranged systematically, with the eponyms generally attached to them and bibliographic references (many British and American). The second is an alphabetical index of syndromes, main symptoms, and eponyms.

The Zollinger-Ellison syndrome, for example, is described in the first part under 'Digestive System', and can be tracked down in the second part by turning up 'Zollinger', 'Peptic Ulcer', etc. However, not all conditions listed in the second part are described in the first.

The accounts of the various diseases are admirably succinct and informative and on the whole well up to date.

A well-produced, interesting, and useful little work of reference, although incomplete without the First Series to which it is complementary. A similar work in English would be welcome.

Transplantation of Tissues. Vol. II. Edited by Lyndon A. Peer, M.D., with twelve contributors. 10 × 6¾ in. Pp. 690 + xiv, with 252 illustrations. Baltimore: The Williams and Wilkins Co. (London: Baillière, Tindall & Cox.) 160s.

To-day can be said to be an era of transplantation because the research worker and the surgeon have gone forward in a united effort to the satisfactory transplantation of human tissues. There is transplantation of skin, cornea, fat, nerves, teeth, blood-vessels, endocrine glands, organs, peritoneum, and cancer cells.

This second volume is ably edited by Dr. Peer, assisted by some thirteen distinguished contributors who are all experts in their own particular field of research. It is refreshing to see the name of Professor P. B. Medawar among the contributors. The pattern of presentation is the same as the first volume which appeared in 1955. To the modern surgeon this second volume, with its clinical applications for research, will make a stronger appeal.

The whole work is a comprehensive survey of an important problem to-day.

Office Orthopedics. By Lewis Cozen, M.D., F.A.C.S. Third edition. 9 × 5¼ in. Pp. 430, with 321 illustrations. 1959. London: Henry Kimpton. 70s.

The third edition of this useful monograph sets out just those conditions which can be treated satisfactorily in the consulting room, and no attempt is made to describe orthopædic conditions which must be treated in hospital.

This book is well illustrated and should prove of real worth to house surgeons, casualty officers, and registrars. However, the price of the book is high and well beyond the purse of most resident medical officers in England.

Surgical Service Guide. By Louis T. Palumbo, M.D., M.S., F.A.C.S., Chief, Surgical Service, Veterans Administration Center, Des Moines, Iowa, U.S.A. 7¾ × 5 in. Pp. 208, with 22 illustrations. 1959. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 45s.

This is a surgical pocket book giving details of pre- and post-operative instructions, together with a useful appendix containing a list of diets, drugs, and hormones. It should prove of value to house surgeons.

The Surgeon and the Child. By Willis J. Potts, M.D., Professor of Pediatric Surgery, Northwestern University Medical School, Chicago. 9¼ × 6 in. Pp. 255 + x, illustrated. 1959. Philadelphia and London: W. B. Saunders Co. 52s. 6d.

This is a small monograph based on the surgical conditions which may occur in infants and children. Whether there is room for the specialized child surgeon or not is a moot point, for in England all the operations described in this book would be performed by general surgeons. This book will prove of more value to the senior student and the general practitioner than to the average surgeon.

A Textbook of Surgical Physiology. By R. Ainslie Jamieson, M.B., F.R.C.S.Ed., Surgeon, Vale of Leven Hospital, Alexandria, Dunbartonshire; and Andrew W. Kay, M.D., Ch.M., F.R.C.S.Ed., F.R.F.P.S.G., Professor of Surgery, University of Sheffield. 9¼ × 5¼ in. Pp. 623 + viii, with 186 illustrations. 1959. London: E. & S. Livingstone Ltd. 55s.

This is an important book and one which surgeons have been awaiting a long time.

The surgeon of to-day requires such a reference book to keep him *au fait* with all the modern advances in the physiological and pharmacological schools of thought. The importance of fluid balance, the endocrine glands, vitamins, and the great advance in our knowledge of the circulation under various conditions are all subjects of surgical physiology.

This text-book is written by two physiologically minded surgeons and they have done their work admirably.

There can be little doubt that this book will become the text-book of choice for all those who are preparing for the Final Fellowship examination; it should also prove popular to candidates who are taking the Primary Fellowship examination.

The book is dedicated to Professor Illingworth of Glasgow and we think he should be proud of his former students and lecturers.

We consider the price of the book most moderate for the wealth of material it contains.

Color Atlas and Management of Vascular Disease. By WILLIAM T. FOLEY, M.D., F.A.C.P., and IRVING S. WRIGHT, M.D., F.A.C.P. 10 × 7¼ in. Pp. 170 + xiv, fully illustrated. 1959. New York: Appleton-Century-Crofts, Inc. $18.00.

THIS is a book in which there are 194 coloured illustrations depicting different forms of vascular disease as seen in 94 patients. There can be little doubt that the understanding of colour changes is so important to the accurate diagnosis of vascular disease.

This book is a reference volume of prime importance and should be in every medical library.

A Text-book of X-ray Diagnosis. Vol. IV. Edited by S. COCHRANE SHANKS, C.B.E., M.D., F.R.C.P., F.F.R., Director, X-ray Diagnostic Department, University College Hospital, London; and PETER KERLEY, C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., D.M.R.E., Director, X-ray Department, Westminster Hospital, London. Third edition. 9¾ × 7 in. Pp. 714 + xvi, with 735 illustrations. 1959. London: H. K. Lewis & Co. Ltd. 110s.

THE new fourth volume of the third edition of this important work deals with the bones and joints and soft tissues and is a most comprehensive survey from the radiographic point of view. With the annual increase of skeletal injuries due to this mechanical age, it behoves the general practitioner to be *au fait* with the common bone injuries and to be able to recognize fractures from radiographs. This fourth volume should prove of real value as a reference book not only to practitioners but to all those who have to treat skeletal injuries.

The X-ray pictures throughout this volume are of a high order of excellence, and the whole work a great credit to British medicine.

Adénomectomie Endo-urétrale. By RAYMOND DENIS. 10 × 6½ in. Pp. 194, with 100 illustrations. 1959. Paris: Masson et Cie. 2500 fr.

THIS is an excellent monograph dealing with all aspects of transurethral resection of the prostate, together with a section on the treatment of carcinoma of the gland. The author is careful to consider all the small but important details concerning the technique that the beginner can seldom find described in text-books. Because of this, it should be possible for him to learn from this book most of the difficulties and dangers of the method, together with the steps that must be taken to ensure good results with few complications.

There are good illustrated descriptions of both Barnes's and Nesbit's methods of resection. The former is preferred for the smaller glands. A large section of the book is devoted to the possible complications, their cause, and the means of correcting them. This is very useful. It is surprising to find no mention of the use of glycine as the irrigating fluid; a metal catheter with two channels is recommended for post-operative irrigation rather than the two-channel Foley catheter. It is pleasing to find stress placed on closed catheter drainage in a French urological book; in so many clinics the catheter is still left hanging into a urinal placed between the legs!

The drawings illustrating the text are excellent. They really assist the reader of the book, which is so seldom the case in books dealing with this subject. It is a pity that many names are misprinted and many references inaccurate.

Lokalanästhesie und Lokalanästhetika. Edited by Prof. Dr. HANS KILLIAN. 10½ × 6¾ in. Pp. 770 + xx, with 341 illustrations. 1959. Stuttgart: Georg Thieme. DM154.

IT is fashionable—and with undoubted justification—to acknowledge the contribution of modern anæsthesia when appraising recent great advances in surgery. One only too readily assumes, with understandable encouragement from our anæsthetist colleagues, that the adjective 'modern' refers exclusively to general inhalation anæsthesia. The publication of this encyclopædic work rescues local anæsthesia from a comparative, quite ill-deserved obscurity.

It is, after all, applicable to practically every branch of surgery, and in much of operative obstetrics at any rate it has recently tended to displace general anæsthesia because of its greater safety.

The subject is treated systematically, and clinical considerations, presented in great detail, are preceded by sections devoted to basic neurophysiology, physical chemistry, pharmacology, and toxicology. Perhaps the only really stimulating part of the book is the study of the general effects of local anæsthetic agents in the human: such concepts as 'endo-anæsthesia' and 'central analgesia' might well receive much greater attention.

Though written in a foreign language, this admittedly costly book can be recommended as a first-class work of reference.

Atlas of Tumor Pathology, Section VII, Fascicles 27 and 28. **Tumors of the Pancreas.** By VIRGINIA KNEELAND FRANTZ, M.D., Professor of Surgery, College of Physicians and Surgeons, Columbia University. 10½ × 7¾ in. Pp. 149, with 92 illustrations. 1959. Washington, D.C.: Armed Forces Institute of Pathology. $1.50.

THIS new fascicle dealing with tumours of the pancreas is as excellent as its predecessors making up the *Atlas of Tumour Pathology*, published by the Armed Forces Institute of Pathology.

The photomicrographs are admirable and could not be improved upon; they are beautifully reproduced. This *Atlas of Tumour Pathology* should be in every medical library because the work is outstanding and as a reference volume could not be bettered.

The Armed Forces Institute of Pathology at Washington have put the world of medicine in their debt for this truly excellent production.

Mitral Valvulotomy. By HAROLD J. STEWART, M.D., Professor of Clinical Medicine, Cornell University Medical College, New York; and FRANK GLEN, M.D., Surgeon-in-Chief, The New York Hospital. 9 × 6 in. Pp. 244 + vi, with 25 illustrations. 1959. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 80s.

THIS is a useful and practical monograph which deals with the clinical records of some 300 patients who have undergone the operation of mitral valvulotomy. All the patients were operated upon for mitral stenosis in the department of surgery of the New York Hospital—Cornell Medical Centre.

Up to the present time no previous work in medical literature has attempted such a critical evaluation of mitral valvulotomy, and this monograph contains important data concerning follow-up autopsies on a high percentage of patients who died in the years following this particular operation.

The occurrence of chronic cholecystitis and gallstones in cases of mitral stenosis makes for careful assessment of both conditions and the surgeon and his medical partner must decide which condition must be treated first.

This monograph is divided into three parts: Part 1 deals with the results of mitral valvulotomy in the first hundred patients in the years 1951, 1952, 1953; Part 2 is a consideration of mitral valvulotomy in the second and third hundred patients in the years 1954, 1955, and to April, 1956; Part 3 is devoted to a critical examination of the long follow-up of the first hundred patients who had mitral valvulotomies.

This work reveals the prime importance of the close association between the physician and surgeon in the treatment of mitral stenosis. It will be read with interest by a large number of cardiological surgeons both at home and overseas.

The Medical Annual. A Year Book of Treatment and Practitioner's Index. Edited by R. BODLEY SCOTT, M.A., D.M., F.R.C.P., and R. MILNES WALKER, M.S. (Lond.), F.R.C.S. Seventy-seventh year. 8¼ × 5¼ in. Pp. 620 + xl, fully illustrated. 1959. Bristol: John Wright & Sons Ltd.

IN the seventy-seventh issue of *The Medical Annual* it is announced that Sir Henry Tidy, who has been associated with the Annual for over a quarter of a century, has retired from Editorship, and the volume contains an appreciation of his work during his long association. His place as joint Editor with Professor R. Milnes Walker has been taken by Dr. R. Bodley Scott. Two other well-known names also pass from the reviewing staff—Sir Philip Manson-Bahr and Dr. Stanley Banks.

The opening chapters are in the form of special articles on New Sex Hormone Preparations, Cancer Research, Cross-infection in the Ward, and Fluid and Electrolyte Balance in Practice, respectively. All are of sterling worth, but perhaps the one which captivates the surgical reader most completely is that on Cancer Research, contributed by Dr. Haddow, who has within the compass of some ten pages of text given a clear and beautifully worded appreciation of the present position and of future trends.

The Review of the Year's Work commences in the customary way with the Alimentary Tract, and contains, amongst other contributions, a comprehensive and authoritative account by Professor Goligher of the Surgery of Cancer of the Rectum. Much space is given later in this section to the drastic method of exposure of the lower end of the œsophagus for cardiospasm by means of splitting the lower part of the sternum and retraction of a rib-flap, as practised in Minneapolis, where the exposure is also used for the surgical treatment of duodenal ulcer. It is to be hoped that the inclusion of this technique in *The Medical Annual* will do nothing to popularize it in this country.

As regards the treatment of duodenal ulcer in which vagotomy forms a part, it seems a little out of keeping with accepted standards to include mention of an article (p. 119) the authors of which ". . . lay stress on the fact that the vagus nerve should be visualized and not palpated. They found that this enables them to do a more complete vagotomy" [*sic*].

The contribution on Anæsthesia comes from the pen of Professor Ronald Woolmer, who replaces Dr. Langton Hewer as reviewer, and illustrates the ever-broadening aspect of this speciality. Both the medical (Dr. Wallace Brigden) and the surgical (Mr. Kent Harrison) aspects of cardiac disease are well covered, the latter containing an excellent set of reproductions of illustrations of the anatomy of atrial septal defects described by Lewis and his colleagues, and published in the *Annals of Surgery* two years ago. Peripheral Vascular Disease is authoritively dealt with by Professor Rob, and contains a description of the ingenious ice-cone method of arterial suture devised by Tibbs of Newcastle.

Pædiatrics includes an account of certain aspects of Child Psychiatry by Dr. Kenneth Cameron, and the usual space is given over to E.N.T. Diseases (not labelled Otorhinolaryngology, it is refreshing to note), and Disease of the Eye. The section on Endocrinology is larger than ever—as may be expected—and contains two impressive colour plates illustrating the effect of adrenalectomy for Cushing's syndrome.

As is the traditional policy, no subject is omitted—Gynæcology and Obstetrics, Infectious Disease, Orthopædics, Skin Disease, etc. One of the most interesting sections, however, and one which is of vital interest to all medical practitioners, irrespective of their main interest, is that on Legal Decisions and Legislatures contributed by E. B. McLellan, Esq., Barrister-at-Law.

The claim of the publishers that *The Medical Annual* "continues to maintain its position as the leading review of medical and surgical progress for well over three-quarters of a century" is upheld by the 1959 edition, though the claim may be challenged by those who regret that in most of the sections—surgery being one of them—the references are almost exclusively confined to articles written in the English language.

The Care of Minor Hand Injuries. By ADRIAN E. FLATT, M.A., M.D., F.R.C.S., Assistant Professor of Orthopedic Surgery, State University of Iowa, U.S.A. 9¼ × 6¼ in. Pp. 266, with 109 illustrations. 1959. St. Louis: C. V. Mosby Company. (London: Henry Kimpton.) 71s. 6d.

MINOR hand injuries are important injuries because if proper and adequate treatment is not administered the result may be crippling and the loss of man hours enormous. In this stage of extension of mechanization and mechanical transport hand injuries are all too common. This is borne out by the number of industrial accidents in this country, in spite of every precaution that can be devised.

This monograph deals with every type of minor injury to the hand. Most hospitals to-day are aware of the importance of adequate and immediate treatment of hand injuries, and have instituted a hand clinic to which all injuries affecting the hand are sent.

The author of this book has worked in the emergency Out-patient Clinic of the London Hospital and is, therefore, quite conversant with English methods.

This book should prove of real value to casualty officers and resident medical officers.

Arterial Embolism in the Limbs. By A. L. JACOBS, M.A., D.M. (Oxon.), F.R.C.P., Physician to the Whittington Hospital, London. 9¼ × 6¼ in. Pp. 200 + xii, with 37 illustrations. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 35s.

THIS is a useful book, for it gives a clear clinical picture of embolic obstruction of an artery in the limbs and then discusses the treatment for this condition. The part played by mitral stenosis and auricular fibrillation in the causation of arterial embolism is well portrayed.

One of the main features of this book is the excellence of the illustrations; some of the radiographs depicting arterial blockage are really beautiful: modern arteriography is playing an important role in the accurate diagnosis of the actual block in the artery.

Professor C. G. Rob writes an excellent Foreword to this monograph and states: "In theory, the treatment of choice should be surgical excision because removal of the embolus is the only way in which a normal circulation can be restored, but in practice physicians and surgeons interested in peripheral arterial disease have treated many of their patients without operation."

Dr. Jacobs gives a list of twenty-four main conclusions and all of these contain information of great value to the clinician and especially to surgeons who undertake the care of patients with arterial emboli.

A most useful book at a most reasonable price.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Health Physics Instrumentation. By JOHN S. HANDLOSER. Being Volume I of the International Series of Monographs on Nuclear Energy—Division V. 8½ × 5½ in. Pp. 120, fully illustrated. 1959. London: Pergamon Press. 35s.

The Mediastinum. By TED F. LEIGH, M.D., and H. STEPHEN WEEKS, M.D. 9¾ × 6½ in. Pp. 246 + x, with 290 illustrations. 1959. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 92s.

Lectures on the Scientific Basis of Medicine. Volume VII, 1957-58. 8½ × 5½ in. Pp. 496 + xii, illustrated. 1959. London: University of London, The Athlone Press. 45s.

Peripheral Facial Palsy. Pathology and Surgery. By KARSTEN KETTLE, M.D., and TERENCE CAWTHORNE, F.R.C.S. 9¾ × 6¾ in. Pp. 341, with 127 illustrations. 1959. Oxford: Blackwell Scientific Publications Ltd. £6.

Le Nouveau-Né. By MARCEL FÈVRE. 9¾ × 6 in. Pp. 200, with 28 illustrations. 1959. Paris: G. Doin et Cie. 2500 fr.

Klinik und Praxis der Urologie. By WERNER STAEHLER. Volume II. 10½ × 7½ in. Pp. 863 + xxii, with 641 illustrations. 1959. Stuttgart: Georg Thieme. DM240.

Atlas Aide-Mémoire d'Anatomie. By H. RAVIERE. Second edition. 10 × 7½ in. Pp. 336, with 241 illustrations. 1959. Paris: Masson et Cie. 4000 fr.

Klinische Clinurgie für die Praxis. By O. DIEBOLD, H. JUNGHANNS, and L. ZUKSCHWERDT. 10½ × 7 in. Pp. 184 + v, with 90 illustrations. 1959. Georg Thieme. DM36.

Histonomy of the Cerebral Cortex. By S. T. BOK, Head of the Netherlands Central Institute of Brain Research. 8¾ × 5¾ in. Pp. 434 + x, with 175 illustrations. 1959. Amsterdam: Elsevier Publishing Company. (London: D. Van Nostrand Co. Ltd.) 72s.

Health Physics. Progress in Nuclear Energy, Series XII, Volume I. Edited by W. G. MARLEY and K. Z. MORGAN. 9 × 6 in. Pp. 600, illustrated. 1959. London: Pergamon Press. £5 5s.

Tools of Biological Research. Edited by HEDLEY J. B. ATKINS, D.M., M.Ch. (Oxford), F.R.C.S. (Eng.), Hon. F.A.C.S., Director, Department of Surgery, Guy's Hospital, London. 8¾ × 5½ in. Pp. 183 + xvi, illustrated. 1959. Oxford: Blackwell Scientific Publications Ltd. 37s. 6d.

A Short Textbook of Surgery. By C. F. W. ILLINGWORTH, C.B.E., M.D., Ch.M., F.R.C.S. (Ed.), F.R.F.P.S. (Glas.), Hon. F.A.C.S., Hon. F.R.C.S. (Eng.), Regius Professor of Surgery, University of Glasgow. Seventh edition. 9¼ × 5¾ in. Pp. 547 + viii, with 263 illustrations. 1959. London: J. & A. Churchill Ltd. 45s.

More Medical and Other Verses. By ALEX E. ROCHE. 7¾ × 4¾ in. Pp. 35 + viii. 1959. London: H. K. Lewis & Co. Ltd. 5s.

Napoleon Immortal. The Medical History and Private Life of Napoleon Bonaparte. By JAMES KEMBLE, Ch.M., F.R.C.S. 8½ × 5½ in. Pp. 307 + xii, illustrated. 1959. London: John Murray. 28s.

Les Entretiens de Bichat 1959. Chirurgie-Spécialités. 8⅞ × 6½ in. Pp. 503. 1959. Paris: Expansion Scientifique Française. 1959 fr.

Metabolic Care of the Surgical Patient. By FRANCIS D. MOORE, M.D., Moseley Professor of Surgery, Harvard Medical School. 10¾ × 7¾ in. Pp. 1011 + x, with 142 illustrations. 1959. Philadelphia and London: W. B. Saunders Co. 140s.

Lectures on the Interpretation of Pain in Orthopedic Practice. By ARTHUR STEINDLER, M.D. 10 × 6½ in. Pp. 734 + xviii, fully illustrated. 1959. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) £7 8s.

The British Encyclopædia of Medical Practice. Medical Progress, 1959. Edited by LORD COHEN of BIRKENHEAD, P.R.S.H., M.D., D.Sc., LL.D., F.R.C.P., F.R.C.P.I., F.A.C.P., F.D.S. R.C.S., F.F.R., F.R.I.C., F.S.A., Professor of Medicine, University of Liverpool. 9¾ × 6½ in. Pp. 327 + xxiii. 1959. London: Butterworth & Co. (Publishers) Ltd. £3 5s.

Textbook of British Surgery. Vol. IV. Edited by Sir HENRY SOUTTAR, C.B.E., D.M. (Oxon.), F.R.C.S., Consulting Surgeon, London Hospital; and J. C. GOLIGHER, Ch.M.(Edin.), F.R.C.S.(Edin. and Eng.), Professor of Surgery, University of Leeds. 9¾ × 7 in. Pp. 699 + viii, with 405 illustrations. 1959. London: William Heinemann Medical Books Ltd. 105s.

Disorders of the Temporomandibular Joint. By LASZLO SCHWARTZ, D.D.S., Clinical Professor of Dentistry, Columbia University. 10 × 6¾ in. Pp. 471 + xxiv, with 168 illustrations. 1959. Philadelphia and London: W. B. Saunders Co. 105s.

Surgical Treatment of Bone and Joint Tuberculosis. By ROBERT ROAF, M.A., M.Ch.Orth., F.R.C.S. Ed., F.R.C.S.Eng., Robert Jones and Agnes Hunt Orthopædic Hospital, Oswestry; W. H. KIRKALDY-WILLIS, M.A., M.D., B.Chir., F.R.C.S. Ed., Orthopædic Centre, Nairobi, Kenya; and A. J. M. CATHRO, M.B., Ch.B., Orthopædic Centre, Nairobi, Kenya. 9¾ × 6½ in. Pp. 137 + viii, with 90 illustrations. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 30s. net.

Treatment of Cancer in Clinical Practice. Edited by PETER B. KUNKLER, M.A., M.D. (Cantab.), M.R.C.P., F.F.R., Radiotherapist, United Birmingham Hospitals; and ANTHONY J. H. RAINS, M.S. (Lond.), F.R.C.S., Professor of Surgery, University of London. 9½ × 6½ in. Pp. 821 + xvi. 1959. Edinburgh and London: E. & S. Livingstone Ltd. £5 net.

Treatment of Cancer and Allied Diseases. Volume III. **Tumours of the Head and Neck.** Edited by GEORGE T. PACK, M.D., F.A.C.S., Attending Surgeon, Memorial Center for Cancer and Allied Diseases, New York; and IRVING M. ARIEL, M.D., F.A.C.S., Associate Clinical Professor of Surgery, New York Medical College. Second edition. 10 × 6½ in. Pp. 781 + xviii, with 1028 illustrations. 1959. London: Pitman Medical Publishing Co. Ltd. £11 11s. net.

Speech and Brain Mechanisms. By WILDER PENFIELD and LAMAR ROBERTS. 9 × 6 in. Pp. 286 + xiv, fully illustrated. 1959. Princeton, New Jersey: Princeton University Press. (London: Oxford University Press.) 35s.

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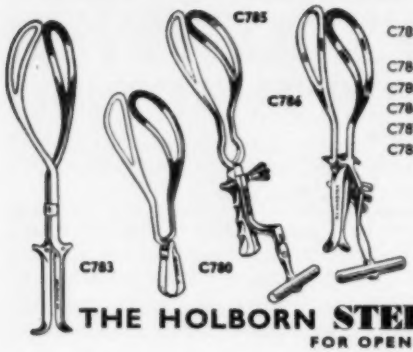

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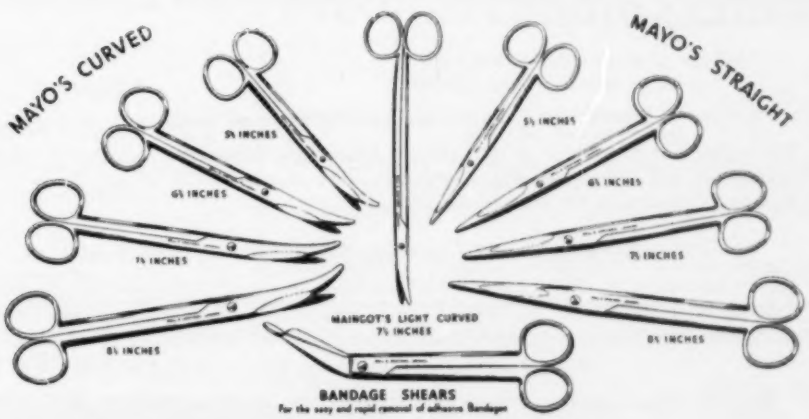

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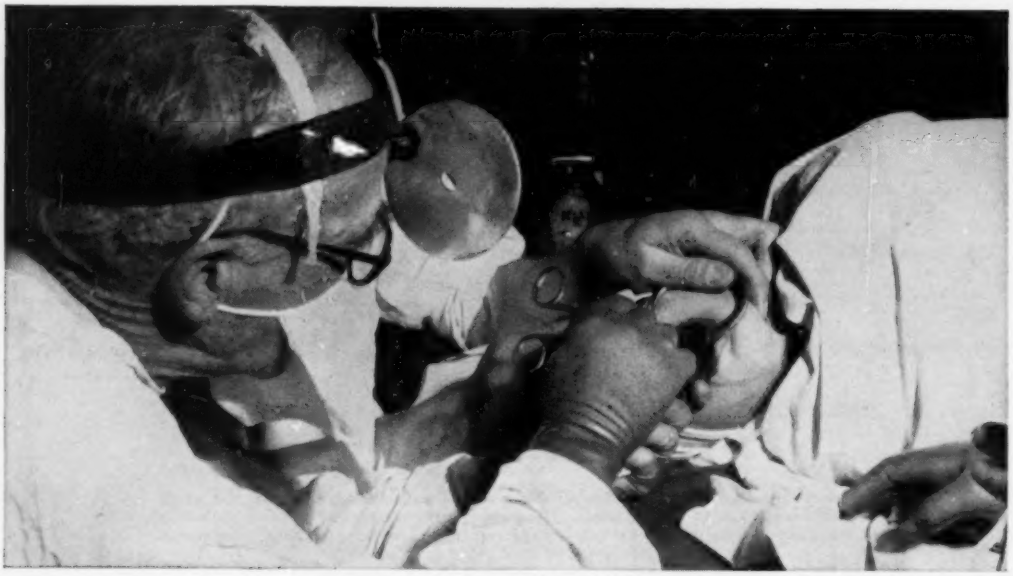

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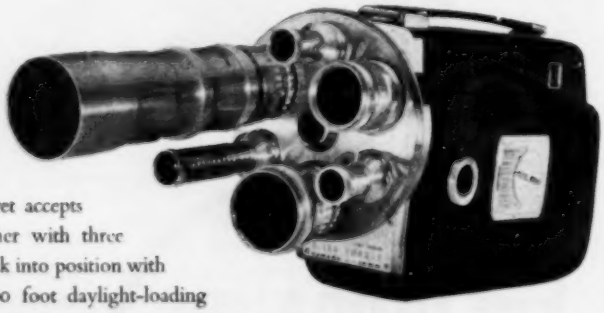

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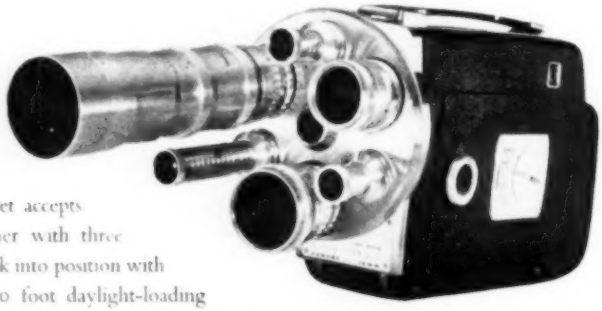

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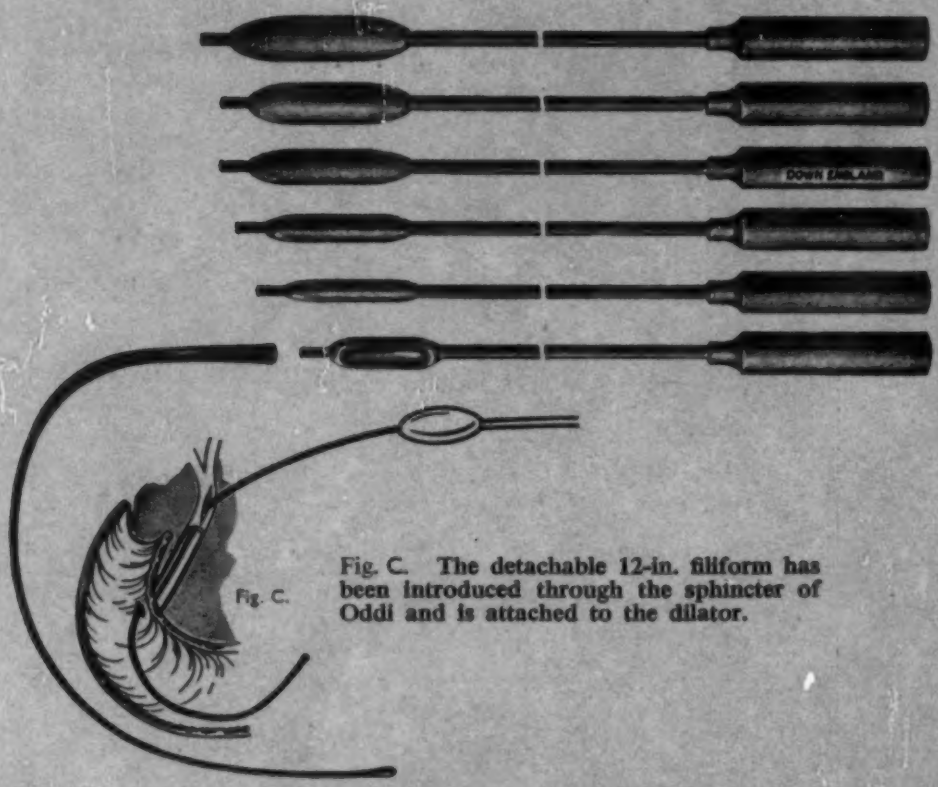

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